DISEASES OF THE CHEST

VOLUME 2

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DISEASES OF THE CHEST

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CHAPTER 1

PLEURISY

KENNETH ROBSON

AETIOLOGY

PLEURISY implies inflammation of the pleural surfaces, whether (1) fibrinous, (2) serous with effusion or (3) purulent The most important single cause of pleurisy, whether acute or chronic, dry or with effusion, is tuberculosis Pneumonic consolidation—whatever the causative organism—single or multiple pulmonary abscess and primary or secondary neoplasia may also be accompanied by pleurisy, as may complicated bronchiectasis Rarely, pleurisy complicates acute rheumatism It may occur in nephritis, often terminally, or during the course of blood diseases such as leukaemia Diaphragmatic pleurisy, with shoulder-tip pain, and mediastinal pleurisy—both localizations with or without effusion—have been described, as isolated phenomena they are rare

It is probable that many of the cases of so-called simple or primary pleurisy are, in reality, due to underlying foci of inflammation in the lung, whilst others are tuberculous The latter aetiology should always be suspected if the pain or rub lasts more than a few days It is very doubtful whether pleurisy ever follows mere exposure to cold—" Pleuritis a frigore " of the older writers Epidemics of primary pleurisy have been described (Scadding, 1946) and are probably to be distinguished from Bornholm disease Pleurisy, evidenced by friction, occurs with varying frequency in the latter, and there is still doubt whether the primary site of inflammation is in the pleura or in the muscles and fasciae of the parietes It is the epidemic, sometimes explosive, nature of an outbreak which stamps it as something distinct In most outbreaks of Bornholm disease, relatively few of the individuals who have the severe, sudden chest pain actually show friction

CLINICAL PICTURE

Pleurisy without effusion

Dry pleurisy whatever its cause, is a painful condition characterized by knife like pain related to the respiratory excursion, which it tends to inhibit locally The pain is not of critically sudden onset, and careful interrogation will generally distinguish it from intercostal neuralgia, fibrositis and pre-eruptive herpes Friction is the cardinal sign and must always be sought The friction has a rubbing, grating or scratching quality and, if coarse, can be palpated with the flat of the hand Pressure with the hand will often cause the pain to disappear, and pressure with the chest-piece of the stethoscope may diminish, or even abolish, the friction sound while pressure is maintained Friction in a peculiar way sounds very " near to the ear " It is often useful to posture the patient so as to stretch the side of the thorax on which the rub is being sought, and to listen exactly over the site of the pain as indicated by the patient With pleural friction, as with other physical

signs in the chest, its absence in a given case by no means excludes the possibility of pleurisy

Radiographic evidence of dry pleurisy is not constant and fluoroscopy only sometimes affords evidence in the form of abnormalities of diaphragmatic function and behaviour X-ray examination, however, will eliminate such conditions as spontaneous pneumothorax, and will help to establish the presence of any intrapulmonary condition of which pleurisy is symptomatic

Pleurisy with effusion

Effusion may develop insidiously or it may follow or be accompanied by pleuritic pain Fluid, in considerable quantities, can accumulate without symptoms if it collects slowly and the patient is thus given time to adjust to its presence gradually On the other hand, it may sometimes accumulate with great rapidity, in which case distress and dyspnoea may become urgent

The physical signs of fluid, whether it be transudate or exudate, pus, chyle or blood, are characteristic and, provided that the quantity is sufficient, unmistakable The apex beat is displaced away from the fluid, movement on the affected side is reduced, and there is decrease in tactile fremitus and impairment of percussion note on the side of the fluid The impairment of percussion note is so striking that the word " stony " has come to be associated with this type of dullness, and eliciting the physical sign may often cause pain in the fingers of the percussor. The breath sounds are absent or altered in strength and may, in certain circumstances, be bronchial Added sounds are no part of the picture unless there be a rub at the upper limit of the fluid The spoken voice often takes on that peculiar nasal quality known as " aegophony " Physical signs are found in the presence of moderate or large collections of fluid, but if the fluid is confined to an interlobar fissure or is less than 150 millilitres approximately in total amount in an adult, it may be unrecognizable by physical examination

Fluoroscopy will show gross collections of fluid immediately The affected part of the thorax is poorly lit and the illumination does not improve with inspiration Turning the patient slowly into the lateral and oblique positions may help to define the extent of the fluid, and the lordotic view will generally show involvement of the greater fissure The x ray plate provides further evidence and a permanent record Fluid gives a shadow as opaque as the heart shadow and diaphragm, with which, according to the amount which is present, it merges A well penetrated view may show its distribution but, despite the older teaching on the subject, the upper limit of the fluid does not invariably run in a gentle curve towards the axilla, and change of posture only slowly brings about re-distribution in the closed pleural cavity, too slowly to permit regular and convincing demonstration of shifting dullness (Figs 1-3, 7*a* and *b*)

TYPES OF PLEURISY WITH EFFUSION

The following are the important groups of causes of a pleural effusion ·

(*1*) *tuberculous effusion* ,

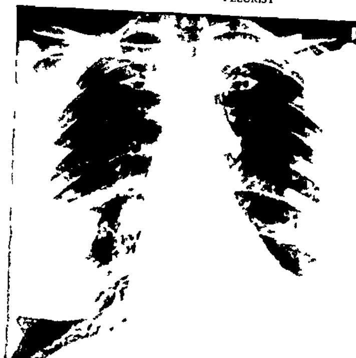
(2) effusion complicating disease in the subjacent lung, other than tuberculosis, of all gradations up to frank empyema ,



FIG 1 — Total left-sided effusion (*By courtesy of the Institute of Diseases of the Chest, Brompton*)



FIG 2 — Left sided lateral effusion (*By courtesy of the Institute of Diseases of the Chest Brompton*)



(a)

Fig 3 — (a) Postero-anterior and (b) lateral views of interlobar effusion on the right side showing opening up of the greater and lesser fissures (*By courtesy of the Institute of Diseases of the Chest Brompton*)



(b)

(3) effusion complicating sub diaphragmatic or hepatic disease
(4) effusion in cardiac and renal disease,
(5) effusion accompanying new growths,
(6) chylous and pseudo chylous effusions

TUBERCULOUS EFFUSION—TUBERCULOUS PLEURISY

To Laennec belongs the credit for the demonstration of the relationship between pleurisy and tubercles in the lung Either dry pleurisy or pleurisy with effusion

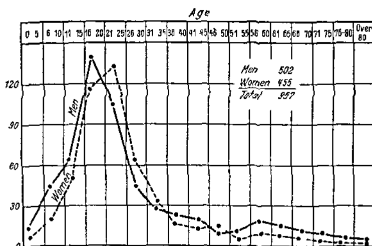


FIG 4—Graph showing age incidence of primary serous pleurisy (idiopathic) (*From Scheel and Foren* 1928)

can occur at any time during the course of pulmonary tuberculosis, and the underlying cause presents no difficulty when established disease with tubercle bacilli in the sputum comes to light in investigation Tuberculous dry pleurisy and more particularly, pleural effusion also occur in the absence of any readily obtained confirmation and in this group the danger of misdiagnosis is very great

Types of tuberculous pleurisy

There are two main varieties of tuberculous effusion

(1) occurring as a late primary manifestation,
(2) due to the presence sub-pleurally of post primary foci

Both groups sometimes continue to a tuberculous empyema The second occurs perhaps more usually in middle aged or elderly subjects and is generally very chronic

A late primary manifestation

Occurring as a late manifestation of the primary infection, tuberculous effusion has its greatest incidence between the ages of 15 and 25 years (Fig 4)

Pleurisy as an allergic phenomenon —The view that the outpouring of fluid represents an allergic phenomenon is maintained by Wallgren (1930) and Rich (1946) An ind [illegible] experienced his primary infection with tubercle bacilli, [illegible] years in response to the irritation of [illegible] its development, with the discharge [illegible] into the pleural space The source of bacilli may also be tub [illegible] onchial adenitis (Erwin, 1944) The degree of hypersensitivity developed varies with the constitution of the individual patient The effusion may be of sudden onset or it may develop insidiously It may be associated with acute symptoms or it may be a casual discovery The latter fact suggests that certain individuals pass through an effusion of limited extent without its ever coming to clinical recognition Proof of this is difficult and an exact expression of its frequency equally so Some idea may be obtained from figures supplied by Brooks Of some 420,000 individuals between the ages of 15 and 75 years examined in the course of a fluorographic survey, 56 cases showed radiographic evidence of recent primary tuberculosis with a symptomless pleural effusion The opportunity arose to observe 63 out of 259 individuals who showed radiographic evidence of recent primary tuberculosis, and in 6 cases a pleural effusion developed within 6 months

Thompson (1946) found that the effusion tended to form on the same side as the pulmonary or glandular lesion There is general agreement that the tuberculous primary complex is right-sided more often than left sided (Kayne, Pagel and O'Shaughnessy, 1947) and in most cases the initial effusion is on the right side

Post-primary effusion

Either dry pleurisy or pleurisy with effusion may occur at any time during the life-time of a consumptive patient Dry pleurisy is often evanescent and marks advance or extension of disease Effusion may develop and, when it does so, the accumulation of fluid is often gradual and insidious The older the patient, the more likely is the effusion to be loculated, the more gradual the accumulation, the slower is the absorption so that it is typical of tuberculous effusion in middle aged subjects to pursue a chronic course, which may, however, eventually have a satisfactory outcome Nevertheless it carries a higher mortality correspondingly than does the juvenile variety The constitutional disturbance may be less severe and the fluid more often blood stained than in the juvenile type The cytology and cultural characteristics are otherwise essentially similar in the two varieties The age incidence of the post-primary effusion, as might be expected, shows a broader base than does the primary type, being similar, in fact, to the age incidence in pulmonary tuberculosis as a whole

Clinical course

Perhaps most typically the primary effusion proclaims its presence with malaise and discomfort in breathing The course of the illness is generally favourable

The temperature subsides in the space of 2 weeks or so the fluid is absorbed function returns and the patient recovers Death in the acute phase of the effusion is rare so that opportunities for post mortem examination are not frequent In some of the subjects which have been examined tubercles have been found on the pleural surface and sometimes there has been evidence of dissemination to other organs

Complications

These can be considered in two groups (1) those which are complications of an active tuberculous process and (2) those which are complications of the presence of fluid in the chest

Complications of the tuberculous process

The complications of the tuberculous process are chronicity and dissemination These are of course interconnected and the chronicity of an effusion and the serial involvement of the opposite pleural space often go hand in hand Dissemination may be on a limited scale or it may be widespread resulting in miliary tuberculosis with the picture dominated by meningitis Other sites to which dissemination commonly takes place are the pericardium the peritoneum and the genito urinary system In the unstable and febrile phase of an effusion dissemination and acute miliary spread are ever present dangers

Concato s disease—polyorrhomenitis or polyserositis—in which there is effusion in both pleurae the peritoneum and the pericardium is nearly always of tuberculous character Rare cases are met with in which the condition remains quite unexplained

Complications of the effusion

The fact of the presence of fluid carries with it the risk of secondary infection either endogenous in intercurrent disease or exogenous Pleural thickening and adhesions are not uncommon sequels of uncomplicated effusion and sometimes marked pleural calcification develops in the course of time Mechanical hydrostatic disturbances may result from the fact of the presence of fluid As is mentioned elsewhere redistribution of the fluid by adjustment due to movement within the thorax may take place in the natural course of events or during aspiration If the redistribution is rapid changes may ensue in the aeration of large districts of the sound lung or in the pulmonary circulation with resulting oedema of the lungs The gravity of either of these events or of a combination of them cannot be over emphasized

Prognosis

Any individual with a hemithorax half filled or more with fluid is in danger on this account alone It is true that the danger is not great but the potentialities constitute a definite threat to life The more immediate prognosis in general terms has been well summarized (Wood 1946) as depending not so much on the extent of the infection as on the unpredictable liability at any stage to acute miliary dissemination

The more remote prognosis has always been a matter of interest and speculation It is said above that a patient recovers But he is a marked man and whether

the illness has been short and sharp or long and smouldering, whether there has been total effusion or a small one, whether serial involvement of one pleural space after another has occurred with recurrences of fever or not, patients who have been through an effusion of this sort have a 25 per cent chance of developing clinical pulmonary tuberculosis within 5 years It was the inescapable fact, of the toll in tuberculous morbidity following pleural effusion, which gradually compelled the adoption of the view that all primary pleurisies are of tuberculous origin unless clearly proved otherwise

Much laborious work has been put in at follow-up inquiries, but there are few series of cases which are free from some sort of objection It is important that a

TABLE I

FOLLOW-UP STUDIES OF TUBERCULOUS PLEURISY

Authors	Percentage of total which are untraced	Size of actual series (cases)	Method of diagnosis	Use of x ray		Observation times		Follow up	Morbidity from tuberculosis
				During attack	Follow up	Minimum	Maximum		
Hedges (1900)	59	130	Needling (67 cases) Phys signs (63 cases)	No	No	Years 2	Years 8	Personal examination	43%
Allard and Koster (1912)	*43 2*	*514*	*Not stated*	*No*	*No*	*2*	*28*	*Mainly* personal examination	*47 7%*
Scheel and Foien (1928)	15	812	Not stated	No	No	1	10	Notification	22 4%
Gaarde (1930)	Not stated	126	Not stated	Partial use		1	12	Partial personal	40%
Borelius (1933)	15	194	No clear information	Not available	Yes	7	17	Personal examination mainly	39 8% 66 3%
Kallner (1937)	15	580	X ray (76%) Clinical (24%)	Yes (76%)	Yes	6	20	Personal examination	39%
Brompton Hospital (1939)	*30*	*111*	*Needling* (76 cases) Clinical and x ray (35 cases)	Yes	Yes	5	12	Personal examination	23% (42%)
Thompson (1946)	Single case (?)	190	Needling (125 cases) Probably all x rayed	Probable	All except 19 cases	1	8	Majority personal	25%
Sinding—Larsen and others (1947)	20 2	474	Not stated	Not stated	Not stated	5	19	Notification	Pulmonary 6 4% All forms 12 4%
Finkler (1947)	33	548	X-rays Some by needling	Not stated	Not stated	5	25	Notification	18% 25%
Press (1947)	Not known	*1192*	*X ray*	*Yes*	Not stated	Not stated		Observation	30%
Maclean (1948)	16 5	183	Not stated	Not stated	Not stated	3	5	Notification	20 5%

series should comprise as large a number of patients as possible and that the number untraced should be minimal Strict criteria are necessary in diagnosis at the time of the effusion (in order to eliminate cases of pulmonary tuberculosis of outspoken type), and the follow-up examinations should be as comprehensive as possible The period of observation should be at least 5 years

The accompanying table summarizes some of the more important facts to be obtained from certain of the published follow up inquiries Each individual series comprises more than 100 cases

Sampling the fluid

When fluid is present in amounts sufficient to give rise to physical signs, it is always wise to take a sample , this is done to confirm the presence of fluid in a doubtful case and to proceed from the recognition of its presence to an attempt to discover why it is there Sampling should always be carried out with full aseptic precautions The best site for exploratory puncture (or thoracentesis) is decided after careful scrutiny of postero-anterior and lateral films, and the puncture is made with a needle mounted on a syringe after infiltration with a local anaesthetic right down to the pleura In an average case the seventh or eighth interspace in the mid axillary line is a suitable site The patient may either be sitting up and leaning slightly forwards with the arms resting on a bed-table, or lying on his side with the site of the effusion uppermost

The fluid is generally straw-coloured (very rarely, if trauma be excluded, haemorrhagic) and clear Sometimes a little clot may form on standing Lymphocytes are abundant and generally predominate, although occasionally even tuberculous fluid is distinctly polymorphonuclear in its cellular content in the earlier phases (Wihman, 1948 , Kraft, 1949) Usually no organism can be seen and culture is sterile on all ordinary media Inoculation into a guinea pig may produce tuberculous lesions in up to 50 per cent of cases, and culture on Löwenstein Jensen's (or Dubos') medium may be positive for tubercle Increasing use will probably be made of cultural methods as techniques are improved and standardized

Close (1946) gives some interesting figures concerning the relative merits of guinea pig inoculation and culture in the detection of tubercle bacilli in pleural fluid, and also of the increasing number of positive results which come with experience Out of 34 tubercle positive fluids, 10 were positive to both guinea pig inoculation and culture, and 24 were positive to culture but negative to guinea-pig inoculation Of 11 attempts to demonstrate tubercle bacilli in fluid, made in 1941, none was successful , in 1945, however, 16 were positive out of 23 There is no doubt that the technique employed is of the greatest importance More recently (Gelenger and Wiggers, 1949 , Calnan and his colleagues, 1951) the glucose content of the fluid has been investigated There is some doubt about the level of glucose which is critical, but low values strongly suggest a tuberculous origin whilst high levels are found in non-tuberculous conditions

Treatment

General management

With tuberculous pleurisy this is a long-term matter In the absence of any reliable specific remedy, the indications are to support the patient by every means

at our disposal while his own recuperative powers and specific resistance develop Rest is the "sheet anchor"—general bodily rest during the febrile phase and stilling of the lung and pleural movement until absorption has taken place Strict rest can be a most valuable measure if the temperature is obstinate in settling, and it is essential in total effusion Patients with large effusions are best nursed propped up and must be very well supported with pillows Just as too rapid decompression of the thorax in these cases can lead to fatal consequences, so can sudden redistribution within the thorax of a large effusion Fidgeting and restlessness can initiate such a redistribution This state of affairs sometimes arises very critically, the aspect of the patient changing in a moment from one of relative comfort and calm to one of alarm, apprehension and respiratory embarrassment He becomes collapsed, cold and clammy, with a rapid pulse and shallow respiration, and it is very difficult to rescue him Oxygen and restoratives should be used at once and aspiration should be carried out (*See also* under "Complications" and "Management of the fluid")

The temperature takes a variable time to subside, sometimes up to 6 weeks From 10 to 14 days after it has become normal provided that absorption is proceeding satisfactorily and there are no complications, a patient may start getting up By the end of a further 6 weeks—that is approximately 3 months from the beginning of the illness—he will have arrived at the stage of being up for 4 hours a day and doing incidental walking of, perhaps, half a mile a day *Graduated walking exercise is now instituted with increased time up, and* by the end of a further 3 months he will be up all day and walking 5 miles per day In most cases it will be permissible at this stage to let him resume work, but supervision and serial x-ray observation must continue for 5 years

A good working rule is to examine clinically and radiographically patients who have had an effusion at the following intervals every 3 months for a year, every 4 months for the second year, every 6 months up to the end of 5 years In the clinical examination, special importance attaches to the weight, and to the possibility that metastatic tuberculous deposits have made their appearance in the interim

Attempts should not be made during convalescence to promote absorption of the fluid or adhesions by expansion breathing exercises or to "unfreeze" a lung (unless after some months of constitutional stability) The aim is a compromise—to obtain settlement of an inflammatory process, which carries with it always the hazard of dissemination Within limits, the anatomical result *per se* is unimportant, and the existence of a poorly expanding hemithorax with movements reduced to a minimum may have to be accepted, at least for a time What should be emphasized is the functional result, as seen by a patient's ability to get about and earn his own living or continue his training Any decision about

Management of the fluid

A sample should always be taken, but it is less easy to lay down rules about

removal of fluid therapeutically The general principle is that so far as possible, the fluid shall be allowed to become absorbed of its own accord Over enthusiasm in aspiration is more likely to do harm than would conservatism even allowing for those patients whose breathing becomes urgently and critically embarrassed There are three indications to interfere

(*a*) in fever lasting a disproportionately long time

(*b*) in fluid persisting a disproportionately long time

(*c*) to relieve respiratory distress

It will often be found that removal of up to 1 pint of fluid will suffice to tip the scale towards stabilization or absorption in the case of (*a*) or (*b*) The method of removal is immaterial provided that the process is slow is conducted under sterile precautions and does not involve the introduction of air into the free pleural space This point of view is maintained although the policy of complete evacuation of the fluid in successive stages and pneumothorax induction is not without its supporters (Feldman and Lewis 1946) For the aspiration a large capacity syringe fitted with a two way tap is convenient Local anaesthesia must be used down to the pleura and a stout needle preferably incorporating some means of clearing with a stilette is the most serviceable Tubing other than stout walled pressure tubing often collapses under suction and aspiration becomes impossible Alternatively Potain s aspirator can be used

When aspiration is undertaken to relieve dyspnoea it is generally because fluid has accumulated rapidly and the circumstances which have initiated the rapid accumulation will in all probability operate again It is unlikely that the removal of a pint or two from a space containing 10 or 12 pints total effusion will contribute much to the comfort of the patient and theoretically the removal of a large amount will be necessary To remove amounts in excess of 3 pints presents technical difficulties

Rapid removal is dangerous involving as it does the risk of disturbing the pulmonary circulation and producing oedema of the lung with albuminous expectoration and death Warning of this development is given by the patients experiencing discomfort and oppression in the chest and by the appearance of

in position connected to an underwater drain or simply to a bottle on the floor by the bedside is very apt to be followed by sepsis and this is a very serious complication indeed Some authors advocate a really big volume aspiration with air replacement compensating for the removal of the fluid by the introduction of air Theoretically this is a sound method but practically it cannot always be carried out and it should certainly not be attempted by the inexperienced Another plan (perhaps the method of choice) is to remove 20–30 ounces daily for 4 6 days although with this method one is very apt to encounter a dry tap at the third or fourth attempt and to fail of one s purpose

Among prophylactic measures are limitation of fluid intake (allowing 30–50 fluid ounces in 24 hours in all) and the administration of diuretics by the mouth

(*Diuretin, 10 grains thrice daily*) or of one of the mercurial preparations by intramuscular injection There are instances in which these measures appear to have been of use

Other methods of treatment

The institution of artificial pneumothorax by air replacement of the fluid is mentioned above The field of application of this technique is, however, probably very limited Pneumoperitoneum with phrenic interruption has its advocates, but insufficient time has as yet elapsed for assessment of its worth as compared with the ordinary conservative regimen

Although there is at present insufficient experience on which to base a final judgment—and here again insufficient time has as yet elapsed for an assessment to be made—there is good reason to think that streptomycin has a definite place in the management of large effusions or those associated with prolonged fever Certainly the immediate benefit is striking The dose for an adult is 1 gramme daily intramuscularly in a single dose The antibiotic is given alone for 7–10 days As soon as the general condition of the patient permits, *para*-aminosalicyclic acid (15 grammes *per diem* in divided doses) is added After a loading dose of 30 grammes of streptomycin in daily injections, the frequency of the injections can be reduced to 3 times a week, perhaps some 45 grammes of streptomycin in all being used

POST PNEUMONIC EFFUSION

This title embraces effusions complicating disease in the subjacent lung, other than tuberculosis, of all gradations up to frank empyema (post-pneumonic effusion)

Aetiology

Empyema thoracis most commonly occurs as a complication of pneumonia, generally pneumoccocal, although any organism may be involved, including *Bacterium coli* and the streptothrix It may also be associated with other infective conditions of the lung, particularly (i) pulmonary abscess, (ii) bronchiectasis or (iii) infected lung and bronchial tissue distal to an obstructing neoplasm What is true of empyema can be regarded as true also of post-pneumonic effusion, which is, in a sense, an attenuated form of empyema The increasing and early use of all forms of chemotherapy in pneumonic illness has led to considerable modifications in the form of pneumonia and to a probable reduction in the over all occurrence of empyema

Differential diagnosis

It is of the greatest importance to distinguish between a post-pneumonic collection of fluid and one due to tuberculosis The whole management and handling of the case is different, but the distinction is sometimes a matter of some difficulty All too frequently a case only comes under close scrutiny after the fluid has formed, perhaps about the tenth day of the illness It will be known that there have been some days of illness, but in all likelihood no information will be available about the leucocytosis and no systematic observation of temperature may have been carried out Some of the differentiating points are now formulated

Tuberculous effusion	*Post pneumonic effusion (attenuated empyema)*
Patient commonly 10–25 years of age	Any age or sex
Pain often early	Pain not an essential feature, especially early
High fever, not responding to sulphonamides or penicillin	May be high fever responding to chemotherapy
No leucocytosis	Leucocytosis early
Fluid obliteration of space on x-ray film from the start	Clear pleural space early on skiagram
Fluid lymphocytic	Fluid *may* be polymorphonuclear
No organisms seen in the smear, except rarely tubercle bacilli	Coccal organisms *may* be seen
Culture on ordinary media sterile	Culture on ordinary media *may* be positive for streptococci or pneumococci
Special culture and guinea-pig inoculation may be positive for tubercle	Negative for tubercle
Prolonged course with slow absorption	Often absorbed within 3–4 weeks

Treatment

The treatment of post-pneumonic effusion, if the disease is of moderate extent, consists in the evacuation of the fluid by aspiration Small collections of fluid may be allowed to be absorbed spontaneously Observation with radiography or under the fluoroscope is necessary to verify that absorption is taking place, and the final check should be made one month after absorption appears to be complete, before the patient is released from observation If the fluid is considerable in amount, it is a good plan, after aspiration, to introduce penicillin intramuscularly If there has been time to do sensitivity tests on the organisms grown from a culture of pleural fluid, then a different chemotherapeutic agent may have been found appropriate and this should be used Penicillin, up to 1,000,000 units, should be injected after aspiration, and as soon as possible expansion exercises should be initiated This active rehabilitation is in sharp contrast with what is practised in tuberculous effusion, but it is of the greatest importance, since re-expansion of the underlying lung is the only satisfactory way to obliterate what is potentially an empyema cavity

EFFUSION COMPLICATING SUB DIAPHRAGMATIC OR HEPATIC DISEASE

Pleural effusion occurs as part of the general dropsical process when cirrhosis of the liver has reached an advanced stage Occasionally the effusion may be the [illegible]

primary tuberculous pleurisy, but sometimes the reaction is strongly polymorphonuclear A careful history may be of the greatest use, supplying evidence of a severe upper abdominal catastrophe within recent weeks Many years of good health may have intervened between the original symptoms of amoebiasis and the development of hepatitis The induction of an artificial pneumoperitoneum may assist in differentiation, since those conditions which are associated with a sympathetic effusion generally bring about adhesion between the liver and the right side of the diaphragm

EFFUSION IN CARDIAC AND RENAL DISEASE

The collection of fluid in the pleural space in cardiac and renal disease is referred to as hydrothorax—a term which serves to differentiate the condition from that in which the fluid results from inflammatory lesions of the pleura In cardiac and renal conditions, the fluid is part of the general dropsical process and the treatment is that of the underlying state In renal disease, subacute nephritis with oedema, both pleural spaces are usually involved, but in cardiac disease, congestive heart failure, the right side may be affected alone This may be related to pressure on the azygos vein by the enlarged right auricle Aspiration may be required, particularly in heart failure The fluid in hydrothorax has a low specific gravity (generally less than 1 015), and only small amounts of protein and a few cells are present, in contrast with the high protein content and cellularity of inflammatory effusions

EFFUSION ACCOMPANYING NEW GROWTH

New growths in the thorax, whether primary or secondary, innocent or malignant, may be accompanied by pleural effusion Effusion is usually not an important part of the picture in innocent growths of the lung or chest wall , but it may be the presenting feature in primary malignant pulmonary disease or the first evidence of intrathoracic metastasis in breast cancer The poorly differentiated form of bronchial carcinoma the so called oat cell type, is more likely to present in this way than is the squamous variety The effusion is an index of the proximity of the growth to the pleura or of massive involvement of root and mediastinal glands by the growth , perhaps, also, it is indicative of the poorly formed vascular channels which characterize a rapidly growing undifferentiated neoplasm

The effusion is often frankly blood stained, even at the very first puncture This is also true of secondary neoplasia, for instance from carcinoma of the breast, with which the pleural surface is studded Cells, showing mitotic figures and with other features suggesting that they may have become detached from malignant growths, may be found in the fluid In order to obtain such evidence it is particularly worth while to examine sections prepared from strands of blood clot and other debris remaining in the lumen of the needle or adhering to its end after aspirations of a few ounces of the fluid

Effusion develops in connexion with benign or malignant growths of the pleura, whether primary or secondary Primary malignancies are excessively rare, but cases are on record, some of which are claimed to be carcinoma and others sarcoma

At the present time it seems fairly certain that some of the cases should be regarded as endothelioma (Buxton and Willcox 1950) Other tumours of the pleura which may be accompanied by effusion are lipoma and fibroma which are benign whilst in addition to the breast the stomach and oesophagus are sometimes primary sites of growth from which metastasis takes place to the pleura Mediastinal dermoid neoplasia in which malignant change has taken place, is sometimes accompanied by a pleural effusion Tinney and Olsen (1945) dealing with 141 examples of pleural effusion due to neoplasms give the distribution of primary sites of origin as shown in Table II

TABLE II

NEOPLASIA CAUSING PLEURAL EFFUSION

Site of primary growth	No of cases	Percentage
Intrathoracic	51	36
Bronchial carcinoma	47	—
Mediastinal carcinoma	4	—
Breast	42	30
Ovary	12	8
Kidney	6	4
Stomach	5	4
Colon and rectum	4	3
Lower urinary tract	4	3
Oesophagus	1	1
Thyroid	1	1
Jejunum	1	1
Not established	14	9

In Meigs syndrome serous pleural effusion sometimes with ascites is found in association with fibroma of the ovary (Meigs and Cass 1937) Since removal of the fibroma is curative an exploratory laparotomy is indicated even when the case appears to be one of inoperable ovarian malignant disease with pleural metastasis

CHYLOUS AND OTHER MILKY EFFUSIONS

Chylothorax is the name applied to an effusion containing chyle the exudate appears as milky fat-containing fluid About 33 per cent of cases follow trauma (MacNab and Scarlett 1932) and of the remainder malignant neoplasms involving the thoracic duct and its tributaries, are the commonest cause but especially in the tropics filariasis is also a cause

The diagnosis is made from the milky appearance of the pleural fluid its oily nature being established by staining with Sudan III Olsen and Wilson (1944)

suggested that the condition was more common than is usually thought In certain cases of reticulosis, tuberculosis and carcinoma in which pleural effusions develop, the fluid may have a milky appearance and contain fat, although it does not contain chyle, such effusions have been described as chyliform Pseudo chylous effusions also occur in which the milky appearance is not due to fat but to other particles causing opalescence, such as a lecithin globulin complex, calcium phosphate, cholesterine or filarial embryos These effusions occur rarely in the reticuloses, in tuberculosis and in neoplastic conditions, and sometimes also in heart or kidney disease

The prognosis in all these cases is grave, the seriousness depending on the underlying condition The best results may be expected in the less severe traumatic cases The effusions tend to recur and, in the case of true chylous effusions, aspiration is not advisable except for diagnostic purposes, because the loss of fat, which results, may be serious

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CHAPTER 2

EMPYEMA

OSWALD TUBBS

NON-TUBERCULOUS EMPYEMA THORACIS

STRICTLY speaking the term, "empyema thoracis", should be confined to the description of a localized collection of pus within the pleural cavity The early stages of bacterial invasion of the pleural cavity and the consequent formation of fluid which is not localized by adhesions must then be described by some other expression, such as generalized or diffuse suppurative pleurisy with effusion However, in this chapter, in order to conform with common practice and for the sake of convenience, the term, empyema, will be used to cover all stages of suppurative pleurisy

ACUTE EMPYEMA

Aetiology and pathology

Because pneumonia is the most common precursor of pyogenic infection of the pleural space, there is a tendency to forget the many other conditions which may lead to suppurative pleurisy

Infections derived from the lungs

When the lung is the source of the infection, it is always possible that some lesion other than pneumonia—such as bronchogenic carcinoma with secondary suppuration, lung abscess, bronchiectasis or actinomycosis—may be responsible for infecting the pleural cavity Owing to the great increase in the incidence of carcinoma of the bronchus, it is particularly important to remember that this dread disease is commonly complicated by an empyema Furthermore, when pneumonia is the cause of the pleural infection, the pulmonary lesion is often not of the classical pneumococcal or streptococcal type for example, an empyema is not an uncommon complication of staphylococcal pneumonia in infants and small children, and is often preceded by a spontaneous pneumothorax, due to the rupture of a small subpleural abscess The pleural space may also be infected from the lung to which the bacteria are carried by the blood stream as a result of a generalized pyaemia or septicaemia, but these cases are now rare owing to the widespread use of chemotherapy

Before the introduction of the antibiotic drugs, pneumonia was frequently complicated by empyema, but there has been a very remarkable decrease in the incidence of this complication during recent years

Infections not derived from the lungs

The infection causing an empyema, however, is not always derived from the lung Bacteria may be carried into the chest by a penetrating wound, but *suppuration is not common unless foreign* matter is retained within the thorax, organic material such as clothing being more likely to lead to infection than

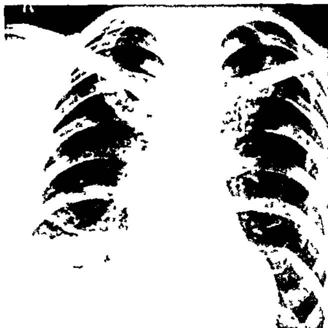


FIG 5 —(a) Right sided subphrenic abscess with a fluid level (b) a total pyopneumothorax after transpleural aspiration of the subphrenic abscess

(a)



(b)

are metallic foreign bodies However, accidental penetrating wounds of the thorax are not common in peace time, but the enormous increase in transpleural surgical operations, such as those on the lung, heart, oesophagus and sympathetic chain, have inevitably led to the surgeon's being responsible for a certain number of cases of empyema, although, with proper post-operative care, the incidence of pleural suppuration following such operations is very low Other sources of infection of the pleural cavity include subphrenic abscess (Fig 5), lesions of the oesophagus, and osteomyelitis of the ribs or spine According to Harley (1949), who reviewed 182 cases of subphrenic abscess, empyema occurred in 29 per cent Diseases of the oesophagus are not frequently complicated by an empyema, because the common lesions of this organ, such as carcinoma, are chronic, thus they allow time for the development of peri oesophageal adhesions, which form a barrier to infection of the pleural space

Sex incidence

Because respiratory infections are more common in males, it naturally follows that pleural empyema has a similar sex incidence

Bacteriology

The bacteria most commonly responsible for pleural suppuration are (1) pneumococci, (2) haemolytic streptococci and (3) staphylococci, in that order of frequency A mixed flora of anaerobic organisms is usually responsible for those cases of empyema which are secondary to foetid pulmonary suppuration, but these organisms may also be found in patients who have no clinical evidence of lung suppuration In a few cases a pure growth of anaerobic streptococci is obtained from culture of the pleural pus More commonly a non haemolytic micro aerophilic streptococcus is the cause of the infection, particularly in patients suffering from multiple areas of suppurative infection of the lung, most frequently seen as a sequel to a major abdominal operation Occasionally an empyema develops as a result of invasion of the pleural space by coliform bacilli, these cases are almost invariably associated with gastro intestinal disease, such as acute appendicitis *Clostridium welchii* alone may be responsible for an empyema, but this is rare except in time of war, for this infection is usually introduced by a penetrating wound Actinomycosis may give rise to suppuration in the pleural cavity and this possibility should not be forgotten, because culture of the pus will not yield any organisms after 48 hours' incubation and this may lead to a report of " sterile on culture ", the nature of the infection can, however, usually be recognized by the presence of the classical ' sulphur granules " In patients who have received a course of systemic chemotherapy, particularly when one of the antibiotics has been used, it is not uncommon to find that the pleural pus shows no organisms on microscopic examination and is sterile on culture These findings may lead the clinician to suppose that tuberculosis is the most likely cause of the empyema, and to treat the patient according to this erroneous supposition—that is to say, external drainage is avoided and the empyema is allowed to become chronic

Morbid anatomy

The initial response to invasion of a normal pleural space by pyogenic bacteria

is acute inflammation of the two pleural surfaces, which is quickly followed by the effusion of a serous exudate, this is turbid on account of the presence of polymorphonuclear cells Although the inflammatory process is unlikely to be of uniform severity throughout the pleural space, nevertheless, at this stage, the fluid is free in the pleural cavity Subsequently the fluid becomes thicker, as the result of an increase in the number of dead and living polymorphonuclear cells and of a rise in the protein content of the fluid This is usually accompanied by the deposition of fibrin, sometimes in very large amounts, particularly when pneumococci are responsible for the infection In cases due to haemolytic streptococci, there is usually very little fibrin present, for these organisms produce a fibrinolysin which breaks down any fibrin deposit On the other hand, an

of the fluid, the pus usually becomes localized to one part of the pleural cavity, most commonly the lower posterior part, as a result of fibrinous adhesion between the two pleural layers In a few cases the pus may be subdivided into two or more separate pockets, because the lung becomes adherent to the parietes between these pockets (Fig 6) Much more commonly, the fluid pus is held in a number of loculi, which are separated only by a layer of fibrin that can easily be broken up with a finger This loculation by fibrin is particularly pronounced in cases arising from infection of a haemothorax

Occasionally the lung is everywhere in contact with the chest wall, and the pus is entirely confined to the space between two lobes However, an interlobar empyema generally extends to the parietes, which means that a portion of the general pleural space is also involved Very rarely the pus becomes isolated to a pocket lying between the lung and mediastinum (" mediastinal empyema "), so that the abscess has no contact with the chest wall It is not strictly accurate to use the term " mediastinal empyema " for cases in which the pus, although largely confined to the space between the lung and mediastinum, also extends to reach part of the chest wall Exceptionally an empyema is entirely confined to the space between the inferior surface of the lower lobe and the diaphragm, and may therefore be described as a diaphragmatic empyema

With the lapse of time, further fibrin is deposited and organization of the deeper layers into fibrous tissue commences Occasionally the pus perforates into a bronchus (broncho pleural fistula) and is expectorated

Clinical picture

Comparative and clinical types

in the course of a streptococcal broncho pneumonia, which was often a sequel to influenza The patients were already seriously ill from the effects of the pneumonia, and infection of the pleural cavity led to a further deterioration of the general condition, marked by distressing dyspnoea and an increase in fever and pulse rate Aspiration of the thin pleural fluid containing haemolytic



(a)



(b)

FIG 6.—Skiagrams showing right sided empyema which was divided into two separate cavities (a) Taken after drainage of a basal para vertebral cavity the upper cavity, which was situated posteriorly and extended into the upper part of the oblique fissure, was subsequently drained by resection of a portion of the eighth rib (b)

streptococci had to be repeated frequently, sometimes more than once a day, owing to its rapid re-accumulation, in order to relieve the patient's dyspnoea Death often occurred and was inevitable if early drainage by rib resection was undertaken

In the metapneumonic cases, the pleural fluid gradually formed after an attack of lobar pneumonia had passed its crisis The pus, which was usually caused by pneumococci, thickened rapidly and soon became localized to one part of the pleural space These patients were not seriously ill but, instead of making a progressive return to health following the febrile crisis, they either continued to run a low grade-fever or, if the temperature had returned to normal, again became febrile about a week after the pneumonic crisis

Because pneumonia is now almost invariably controlled within 72 hours by appropriate chemotherapy, it is no longer practical to divide empyemas into synpneumonic and metapneumonic categories, but this old classification serves to remind the surgeon never to undertake drainage by rib resection until the empyema is localized by adhesions and the pneumonic process completely resolved

Symptoms and signs

Nowadays the presence of pleural fluid following or accompanying pneumonia is usually recognized from routine physical or radiological examination, although severe pleural pain in the early stages of the pneumonia, or persistence of pyrexia in spite of adequate chemotherapy, may have already led the clinician to speculate on the likelihood of this complication It is important to realize that the absence of fever by no means excludes the presence of pus, particularly if the pus is sterile

is usually denoted by a sudden deterioration in the general condition and by increased dyspnoea, but some cases develop remarkably insidiously Occasionally, when an empyema does not receive prompt and adequate treatment, the pleural pus finds its escape within a few weeks by perforating into a bronchus This complication leads to the sudden expectoration of large quantities of pus, but this development is often preceded by a period lasting several days, during which the patient develops an increasingly " wet " cough, due to irritation of the bronchus into which the pus later perforates Just before the fistula is formed, the sputum sometimes becomes slightly blood-stained After the initial episode occasioned by the formation of the broncho pleural fistula, the patient will continue to expectorate pus and usually notices that this is exacerbated by a change in posture

With regard to the physical signs, every student is taught the classical findings of impaired movement, absent tactile fremitus, dullness or impaired note on percussion, absent or reduced breath sounds, and diminished voice conduction

pression collapse of the lung tissue beneath the fluid The position of the mediastinum is useful in distinguishing a pleural effusion from pulmonary atelectasis, but it cannot be relied on to differentiate between the two conditions,

for the mediastinum may not shift towards the opposite side unless an effusion is very large, and collapse of a whole lobe often occurs without causing the mediastinum to move towards the side of the lesion

Radiological appearances

All types of pleural fluid—serous, purulent, bloody and chylous—produce a similar homogeneous opacity on x ray examination of the chest In the earliest stages of an empyema the fluid is free in the pleural space, and its position is influenced to a certain degree by gravity, but to a much less extent than would be expected (Fig 7) With moderate-sized effusions, skiagrams taken with the patient sitting upright show a diffuse opacity at the base, and this often rises highest in the axilla In films taken with the patient supine, the opacity becomes slightly less dense at the base and extends further upwards, but the change is usually not great The skiagrams taken with the patient sitting upright may show a fluid level, but *only* if there is gas as well as fluid in the pleural cavity A fluid level therefore indicates one of three possible alternatives (1) air has been allowed to enter the pleural cavity from the exterior, either through a needle introduced at a previous paracentesis or as a result of a surgical or accidental wound , (2) a broncho-pleural fistula has formed, permitting air to pass from the lung into the empyema , or (3) the suppuration is caused by gas producing organisms (Fig 8) With regard to the last-mentioned, the appearances after infection of a haemothorax by gas-producing organisms, such as *Clostridium welchii*, may be very striking for the gas is prone to become trapped in many fibrinous loculi, giving rise to multiple fluid levels

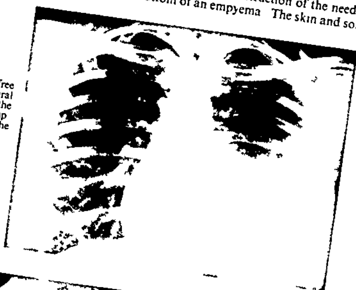
As soon as the empyema becomes localized by adhesions, the radiological opacity remains constant whatever the position of the patient Frontal and lateral skiagrams should be taken, using a penetration above that normally employed for routine chest radiography, in order to show the exact position and extent of the empyema Empyemas of medium or small dimensions are usually located in the paravertebral gutter and extend upwards from the diaphragm , less commonly the pus is localized to the axillary region The uncommon interlobar empyema gives rise in a lateral skiagram to an elliptical shadow in the line of one of the fissures The rare mediastinal empyema appears in a frontal view as an opaque bulge, continuous with the opacity normally produced by the mediastinal structures

Diagnostic aspiration

The diagnosis requires confirmation by the aspiration of a sample of pleural fluid In order to avoid unnecessary discomfort to the patient, it is preferable that the position of the fluid should be demonstrated radiologically before aspiration is attempted

Paracentesis is performed under local anaesthesia, with the patient firmly but comfortably supported in bed If the pus is located posteriorly, the patient may either lean forwards across a cardiac table or sit sideways on the edge of the bed (Fig 9*a* and *b*) If the axilla is chosen as the site for puncture, the patient need not move in bed, except to put the hand of the affected side behind his head so that the arm is lifted out of the operator's way (Fig 9*c*) Because the initial aspiration

is made primarily for d agnostic purposes it is preferable to select an intercostal space wh ch is considered to lie well above the lowest limit of the empyema for attempts to enter the bottom of the cavity may lead either to penetration of the d aphragm which may be higher than expected or to obstruction of the needle by fibrin wh ch tends to gravitate to the bottom of an empyema The skin and soft



F G 7 --Flu d free n the left pleural cav ty (a) W h the pa ent s t ng up r gh (b) w h the pat en recumbent

(a)





FIG 8—Foetid empyema due to gas forming organisms This is not a total empyema as the lung is adherent to the chest wall at the apex

tissues over the selected site for aspiration are infiltrated with procaine solution through a fine hypodermic needle, and the skin is punctured with the point of a scalpel A large bore needle (at least size 17 B W G) is then fixed to the end of a two way adaptor, which is connected to a 20-millilitre syringe filled with procaine solution This needle is introduced into the skin puncture and steadily advanced through the chest wall, simultaneously infiltrating the tissues freely with procaine solution in order to ensure a painless puncture Three test tubes, one of which contains a small quantity of 3 8 per cent sodium citrate solution, should be ready to receive samples of the aspirate , the tube containing citrate is used only if the fluid is found to be serous and therefore likely to clot One of the non citrated specimens is placed in a rack in the ward and the others are sent for pathological examination

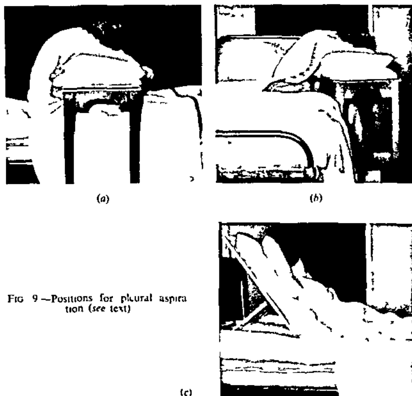
If pus is obtained, it is worth while to continue the aspiration for therapeutic purposes, using the two way adaptor to discharge the fluid into a receiver, thus

pleural space at the end of the aspiration

Treatment

Aspiration and chemotherapy

If the presence of a pleural effusion is recognized early, if aspiration produces fluid from which the solid deposit on standing is less than one third of the total volume, and if the bacteria are sensitive to some antibiotic suitable for intra-



(a) (b) (c)

FIG 9—Positions for pleural aspiration (*see* text)

may entail aspiration on alternate days or even daily At the end of each aspiration the appropriate antibiotic is injected into the empyema and this should not be omitted until it is found impossible to demonstrate bacteria in a film of the pus on at least two consecutive occasions It is more satisfactory to rely on microscopic examination of the pus than on cultures, for bacteria may be present and yet fail to grow when incubated on suitable media In the case of penicillin sensitive bacteria, it is suggested above that the initial dose should be in the region of 500 000 units but, subsequently, it is doubtful whether anything is gained by exceeding 100 000 units at each intrapleural injection

It cannot be over-emphasized that treatment solely by aspiration and local chemotherapy is justified only when the infected pleural effusion is recognized early, and it should not be continued unless the fluid becomes progressively thinner and ceases to re accumulate within 2–3 weeks There is a great tendency

almost entirely from the character of the pus When the pus was thick, so that, on standing, the solid deposit exceeded half the total volume, the empyema was presumed to have become localized by adhesions This was a fair assumption and still applies to-day A sample of pus should therefore be taken at the time of each aspiration and placed in a rack in the ward, so that the amount of deposit on standing may be seen at a glance If local chemotherapy is used, however, the pus may not become increasingly thick, although the empyema has in fact become localized Consequently, evidence that the empyema has become

adhesions In cases of doubt a small quantity of air, but not more than 10 millilitres, may be injected into the empyema, skiagrams being subsequently taken in the upright position, in order to determine whether this air is free to rise to the apex of the pleural cavity or whether, on the other hand, it is restricted to a limited empyema cavity (Fig 11)

All localized empyemas should be drained by rib resection, even if the pus is sterile and the patient apparently free from toxaemia Before doing so, 5 millilitres of iodized oil are injected into the cavity and frontal and lateral skiagrams taken, using increased penetration The oil falls to the bottom of the empyema, and it is therefore possible to determine from the skiagrams the exact site at which

oil falls below this level, because it is claimed that drainage is likely to be hindered by a rise in the position of the diaphragm following operation It is certainly uncommon for the oil to drop below the levels mentioned, but, when it does do so, there should be no hesitation in resecting a portion of rib at the level demonstrated as the bottom of the cavity, for the tube can always be lengthened after operation if the diaphragm rises

Technique of rib resection and external drainage

The operation is performed under local anaesthesia with the patient lying in that position which provides the surgeon with the most easy access to the site selected for drainage *For resection of a rib posteriorly or in the axilla*, the patient lies on the " good " side, buttressed by a large support placed anteriorly An operation should never be performed until the patient is fit enough to lie comfortably in this position for as long as is required This does not apply to *cases complicated by a broncho-pleural fistula*, because operation must then be performed with the patient firmly supported in the sitting position, for recumbency is likely to cause the pus to enter the bronchial tree through the fistula and thus promote incessant coughing or even drown the patient

An incision 3–4 inches long is made over the portion of rib selected for resection, ... If the resection ... along the lateral ... in all other cases, the incision is preferably made in the line of the rib, although some surgeons



FIG 11 —Frontal and lateral skiagrams of a localized empyema Iodized oil has been injected to show the bottom of the cavity Air in the upper part of the empyema shows that the cavity is limited by adhesions (The air in the cavity was allowed to enter accidentally in this case and far exceeds the recommended maximum of 10 cc)

(*a*)



(*b*)

recommend a vertical incision for all cases At least 3 inches of the rib should be resected subperiosteally A needle is then passed through the rib bed, in order to confirm that the site chosen for operation does in fact overlie the empyema As soon as pus is obtained, a small incision is made through the rib bed so that the pus may escape slowly When the flow of pus diminishes, the opening into the empyema is extended for the full length of the portion of rib resected The rest of the pus is subsequently removed by suction and all fibrinous masses are extracted by means of sponge-holding forceps The empyema is then inspected with the aid of a light suitable for introduction into the cavity, the light on the end of a cystoscope will serve this purpose if there is no special instrument available Any fibrin adhering to the parietes or lung surface should be gently wiped away and extracted The mobility of the lung may be ascertained by getting the patient to cough gently If the empyema has not been entered at its lowest limit, a further portion of the same rib or a segment of another rib must be resected, so that the cavity may be drained from the bottom A large rubber tube—at least ½ inch in internal diameter for an adult—is inserted so that the inner end projects about 1 inch inside the cavity Finally the superficial muscles and skin are sutured around the drainage tube This tube is held in position by transfixing it on a level with the skin with a safety-pin, which is anchored to the chest by long pieces of adhesive strapping The dressings are applied around the tube and held in position with gate strapping Bandages or strapping which encircle the chest should be avoided because they restrict respiratory movement. Unless the empyema is very small, the drainage tube should be connected to a water-seal bottle, in order that the patient may develop a moderate negative pressure within the empyema and so encourage re-expansion of the lung Water-seal drainage also provides for the discharge to be carried away from the dressings (*Fig* 12)

Drainage by an intercostal tube—disadvantages

In this discussion on treatment, the use of intercostal tube drainage has not been mentioned Such drainage should never be employed for an adult in the treatment of a localized empyema, because the introduction of the tube does not provide an opportunity to remove fibrin and the calibre of the tube is necessarily too small to provide efficient drainage Furthermore, an intercostal tube often becomes painful after a week or so, because of irritation of the intercostal nerve, and it sometimes causes pressure necrosis of part of one of the adjacent ribs However, some clinicians maintain that, prior to localization of an empyema, the use of an intercostal tube connected to a water-seal bottle is preferable to repeated aspiration Before the introduction of effective local chemotherapy, it could be argued that continuous drainage through an intercostal tube was better than intermittent aspiration, particularly in those cases of streptococcal effusion which re collected very rapidly But, with the use of antibiotics, it is rare to see an effusion re collect quickly, and most empyemas soon become localized and ready for drainage by rib resection It is therefore doubtful whether there is now any place for intercostal drainage in the treatment of empyema If it is employed it should always be followed by rib resection, although an exception to this rule may sometimes be made in the case of small children

Post operative care

period depending on the size of the empyema and the mobility of the walls of the cavity In the case of large cavities re-expansion of the lung will be assisted



FIG 12—This photograph illustrates water seal drainage the method of fixing the tube to the chest wall and the gate-strapping used for keeping the dressings in position

by the negative pressure induced by water seal drainage provided that the lung is not bound down by rigid scar tissue As a practical guide it is probable that water seal drainage is serving a useful purpose so long as the column of water in the glass tube shows a large respiratory "swing and a mean negative pressure exceeding 10 centimetres With an empyema of less than 500 millilitres capacity at the time of drainage it is usually unnecessary to persist with water seal drainage for more than a week but with large cavities it may be advisable to continue for 2 3 weeks When the water seal is deemed to have completed its purpose the tube is cut off close to the safety pin with which it is anchored to the chest wall and any discharge subsequently drains into the dressings This has the great advantage of allowing the patient to move more freely

It is very important that the patient should perform breathing and postural exercises under the direction of a physiotherapist who is specially versed in this form of treatment The exercises are started on the day following operation they are continued until the empyema has completely healed and further until respiratory movements have been restored to normal The patient should practice these exercises as much as possible throughout the day and not only in the presence of the physiotherapist

Re-expansion of the lung will also occur more quickly if the patient is physically active He should therefore leave his bed early and undertake as much exertion as the general condition permits Most patients are able to get up within 24-48 hours of operation

Irrigation of the empyema cavity which was popular at one time and is still

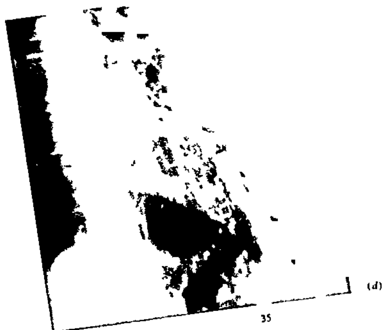
FIG 13.—Serial "sinograms" to show progress in obliteration of an acute empyema cavity. (*a*) The tube is correctly placed at the bottom of the cavity; (*b*) the cavity is slightly smaller. In (*c*) a tube of excessive length has been inserted and a bronchial fistula has developed (probably due to ulceration secondary to pressure from the end of the tube) (*d*) The fistula has healed although some residual oil can be seen in the alveoli The tube is again too long and requires shortening The tube is not removed until the sinogram shows complete obliteration of the intrathoracic cavity.

(*a*)

(*b*)



(c)



(d)

frequently employed, should not be done, for it serves no useful purpose and may be harmful, because there is always a risk that secondary infection will be introduced It is obviously strongly contra indicated if a bronchial fistula is present

Control of the drainage tube

The rapid obliteration of an empyema depends more on the maintenance of perfect drainage than on any other factor Lateral and frontal skiagrams should be taken within 48 hours of surgical drainage A fluid level in the empyema indicates imperfect drainage , this may be due to blockage of the tube by fibrin , in other cases the tube is too long and so projects above the bottom of the cavity, but sometimes it is too short, so that the inner end has been withdrawn from the cavity into the chest wall In any of these circumstances, the tube must receive immediate and appropriate adjustment

The length of the tube is subsequently controlled by frontal and lateral skiagrams, taken after introducing 5 millilitres of iodized oil into the empyema through the tube The oil should be injected with the patient lying down , the tube is then occluded with a spigot and the skiagrams are taken with the patient in the upright position From these ' sinograms " or " pleurograms ", which should be taken at intervals not exceeding 2 weeks, it is possible to determine the relation of the inner end of the tube to the bottom of the cavity (Fig 13) The tube can then be adjusted so as to provide dependent drainage These films usually also show the size of the empyema The tube must not be removed until " sinograms " have shown that the empyema cavity has been completely obliterated This usually occurs 4–10 weeks after operation Large cavities and those drained after the optimal time require the longest period of drainage Patients who require prolonged drainage should be encouraged to return to their employment with the tube still in position

Possible future advances in treatment

One of the strongest arguments in favour of performing rib resection in all cases of localized empyema is that this provides an opportunity to remove all the fibrin from the cavity It is now possible to obtain commercially enzymes which are capable of breaking down within the human body the solid constituents of an inflammatory exudate These enzymes, which are derived from the [illegible] and streptodornase

and Read, 1950) It is possible that the intrapleural injection of these enzymes may obviate the necessity for drainage of some cases of acute empyema but it would be unwise to recommend this as a routine form of treatment until these ferments have received more extensive trial They are likely to be of particular value in empyemas which arise from infection of a haemothorax

During recent years some surgeons have tried to reduce the period of convalescence by first sterilizing the empyema with intrapleural chemotherapy and then treating the residual cavity by decortication (*see* page 44) Complete decortication followed by an uncomplicated convalescence certainly gives quick results but decortication is a major operation compared with rib resection drainage

and is not always followed by complete and rapid re-expansion of the lung It is therefore advisable to reserve this form of treatment for those patients who are seen in the first instance several weeks after the optimum time for external drainage

Empyema complicating diseases other than pneumonia

(1) *Secondary to septicaemia or pyaemia* —In patients with a generalized blood infection, the occurrence of pleural pain, dyspnoea or an unexplained deterioration in general condition should lead to a careful investigation for evidence of infection of the pleural space Fortunately, these cases, which were often bilateral, are now rare, as the result of the widespread use of chemotherapy

(2) *Empyema secondary to subphrenic abscess* —Reference is made above to Harley's review of 182 cases of subphrenic abscess (Harley, 1949), in which he found that empyema occurred in 29 per cent, but that 50 per cent of these cases followed injury to the diaphragm, caused by transpleural drainage, a penetrating wound or transpleural aspiration (Fig 5) On the other hand, he found that a serous pleural effusion occurred in 25 per cent. This means that, if the diaphragm is uninjured, a pleural effusion arising in the course of a subphrenic abscess is more likely to be serous than suppurative, which emphasizes the importance of diagnostic aspiration in these cases A serous effusion will clear up spontaneously provided that the subphrenic abscess is drained satisfactorily An infected effusion should receive treatment similar to that described above for a post-pneumonic empyema When the empyema is localized and ready for external drainage, the site for rib resection should be chosen by the method described above and should be separate from the wound made to drain the subphrenic abscess

(3) *Empyema secondary to bronchiectasis* —If the infection associated with bronchiectasis extends into the lung parenchyma, the patient suffers an acute febrile episode, usually described as pneumonia In some cases, if the pleural space is not obliterated by adhesions, the infection spreads further and gives rise to an empyema This possibility should not be forgotten because the diagnosis is apt to be missed, so that the empyema becomes chronic, and then, if a broncho-pleural fistula develops, the increased expectoration is attributed to an exacerbation of the bronchial infection

(4) *Empyema secondary to lung abscess* —If a foetid lung abscess ruptures into the pleural space, the patient often becomes dangerously ill within a few hours In these cases the injection of penicillin into the stinking pyopneumothorax is often life-saving

(5) *Empyema secondary to bronchiogenic carcinoma* —In patients of cancer age who develop an empyema, the possibility of an underlying growth should always be considered, particularly in males, and if the acute illness was preceded by a considerable period of lassitude or by symptoms of bronchial irritation In cases of doubt the patient should be subjected to bronchoscopy before deciding on treatment of the empyema If a growth is found, the presence of the empyema does not exclude the possibility of successful resection If there are other factors indicating that the growth is inoperable, every effort should be made to control the empyema by aspiration and intrapleural chemotherapy, so that external

drainage may be avoided, for a discharging wound only adds to the miseries of a dying patient

(6) *Empyema due to actinomycosis*—Infection of the pleural cavity by actinomycosis characteristically produces a thin layer of pus with much loculation, but, exceptionally, a single cavity simulating a postpneumonic empyema occurs Infection of the pleura is often followed by much pain and later by induration of the chest wall and sinus formation Drainage operations are rarely required for almost all cases clear up completely and permanently as a result of prolonged and intensive systemic penicillin therapy It is suggested that at least 2,000,000 units should be given daily for a minimum period of 6 weeks

Complications of acute empyema

(1) *Broncho pleural fistula*—Fistulae which complicate an acute empyema almost invariably heal after dependent drainage of the empyema

(2) *Failure of the lung to re expand and obliterate the empyema cavity*—See "Chronic Empyema"

(3) *Infection of the chest wall*—Before antibiotics were used locally to control the infection in empyemas, it was common to see an abscess in the chest wall at the site of a previous paracentesis, but such cases are now rare In a few instances, spreading infective necrosis of the skin has been observed, usually after drainage of the empyema These serious cases should be treated by immediate excision of the affected tissue with the diathermy knife, and the raw area so caused should be subsequently grafted with skin

(4) *Haemorrhage from the intercostal vessels, following drainage of the empyema*—This rare complication may be recognized by the occurrence of persistent and profuse haemorrhage around the tube The tube should be removed at once and the wound packed with a gauze roll The patient is then taken to the operating theatre so that the wound may be re opened and the vessels ligated Some surgeons claim that this complication does not occur if the vessels are routinely divided between ligatures at the time of draining the empyema

(5) *Brain abscess*—Because it is rare for a straightforward acute empyema to give rise to a cerebral abscess, this complication is considered at the end of the discussion on chronic empyema

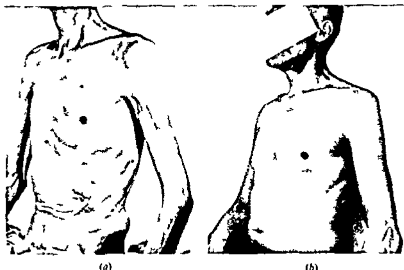
CHRONIC EMPYEMA

It is obvious that all cases of chronic empyema have passed through an acute stage and that the transition from the acute to the chronic stage is a gradual one, so that there is no clear line dividing the two conditions Furthermore the conditions under which an empyema may be reasonably defined as 'chronic" are subject to the personal views of individual clinicians, but it is suggested here that the term, chronic, should be applied to those cases in which a cavity persists for more than 3 months after the original pleural effusion

Aetiology and pathology

If an acute pyogenic empyema is not drained externally or aborted by aspiration and local chemotherapy, more and more fibrous tissue is laid down, particu-

larly on the parietal layer of pleura thus immobilizing the chest wall and preventing expansion of the lung It is necessary to emphasize that the fibrous tissue is laid down on the surface of the pleura and therefore should not be called pleural thickening, although it is often described as such Sometimes the pus finds its escape through a broncho pleural fistula and is expectorated this is mentioned above under Acute Empyema In other cases the chest wall is eroded and some of the pus forms an abscess beneath the overlying skin and superficial muscles —a condition designated empyema necessitatis (Fig 14) this is soon followed



(a) (b)

Fig 14—Empyema necessitatis due to actinomycosis (a) the swelling above and medial to the left nipple is a subcutaneous abscess communicating with an empyema (b) 4½ months later after treatment with systemic penicillin (440 mega units were given over a period of 3 months) therefore no operative treatment was employed

by necrosis of the tissues covering the abscess and the pus escapes externally leaving a sinus which may heal intermittently or discharge persistently

The fibrous tissue formed during this chronic stage gradually contracts so that there is progressive flattening of the chest wall which is accompanied by a scoliosis with the concavity towards the affected side In addition the mediastinum which may have been displaced towards the healthy side during the acute phase is slowly drawn towards the side of the empyema (Fig 15) The chronic inflammation also causes an irritation of the periosteum of the ribs overlying the empyema and consequently new bone is laid down on the inner surface of the ribs so that they become triangular in cross section (Fig 16)

These changes are not confined to persistent undrained cavities for exactly the same series of events occurs when a drained empyema is not rapidly obliterated by re-expansion of the lung The most common cause of persistence of an empyema in spite of external drainage is premature removal of the tube A



FIG 15—Left sided chronic empyema in a girl aged 18 years which started as an acute empyema at the age of 6 years Iodized oil has been injected into the cavity and is just visible at its base



FIG 16—Chronic empyema showing an extreme degree of periosteal new bone formation The cavity has been drained and a tube which is far too long inserted Iodized oil injected through the tube has entered a bronchial fistula and outlined the bronchial tree

residual cavity is also likely to occur if there is any interference with free drainage, as, for example, when the tube is too long or too short, or when the drainage opening is not at the bottom of the cavity Sometimes an empyema fails to heal because the pleural infection is maintained by the presence of a foreign body, such as a piece of drainage material or a sequestrated portion of rib. In other cases the infection may be kept up by an unsuspected aetiological factor, such as tuberculosis, actinomycosis or pleural carcinomatosis A chronic cavity may also persist because the lung is not free to expand This may be due to the presence of intrinsic disease, such as carcinoma, or fibrosis associated with a chronic lung abscess or bronchiectasis More commonly, however, the lung is healthy, but is unable to re expand because it is covered with a rigid layer of fibrous tissue The latter may occur as the result of a delay in draining the empyema or because the surgeon omitted to remove all the fibrin at the time of operation The lung is also likely to become covered with a thick layer of scar tissue if a total empyema has been caused by drainage of a septic pleural effusion before it has become localized by adhesions A broncho-pleural fistula sometimes leads to a delay in re-expansion of the lung but, unless the fistula is very large, it does not often cause an empyema to become chronic, provided that the underlying lung is otherwise healthy

Clinical picture

Clinical types

Two clinical types of chronic pyogenic empyema may be recognized (1) "latent", in which drainage has never been performed, either because the diagnosis was not made during the acute stage or because the clinician did not recognize the need for external drainage, (2) "persistent", in which a cavity persists in spite of external drainage

Latent empyema.—A patient with a latent empyema may be free from symptoms, and may have been referred for treatment because the signs of an encysted collection of fluid have been discovered on clinical or radiological examination This applies particularly to those cases in which the pus is sterile as a result of either local or systemic chemotherapy In other cases the patient complains of persistent fever, chest pain or dyspnoea following an acute respiratory infection Sometimes the patient notices that, after recovering from an attack of pneumonia, he has not returned to the same state of vigorous health which he enjoyed before his illness Then, after an interval which varies from a few weeks to several years, he may suffer an acute febrile episode accompanied by pleural pain If the correct diagnosis is not recognized and appropriate treatment instituted, these symptoms may gradually subside but are liable to recur again and again As long as a latent empyema persists, it may at any time develop into an empyema necessitatis the patient will then complain of a tender swelling in the chest wall

Persistent empyema.—A patient with a "persistent" chronic empyema most commonly complains because there is a continuous or intermittent discharge from the drainage wound following removal of the tube Sometimes the wound heals and remains healed for quite a long time, perhaps for a period of several years, and then becomes the site of an abscess which is, in fact, an empyema necessitatis In other cases of "persistent" empyema the tube has never been

removed, and further treatment is sought because the empyema cavity does not show signs of undergoing progressive obliteration

Broncho-pleural fistula.—A latent empyema or an ill drained persistent cavity may at any time lead to the formation of a broncho-pleural fistula: the most prominent symptom is then likely to be cough with purulent, and sometimes blood-stained, expectoration

All those affected with chronic empyema are prone to suffer from lassitude and other symptoms of poor general health

Signs of chronic empyema

The complexion of a patient with a chronic empyema is frequently pale and "muddy", if the septic process has continued active for more than a month or two Pleural suppuration almost invariably leads to clubbing of the fingers and toes, sometimes within a few weeks of the onset of the infection Physical signs in the chest include gross flattening of the affected side, and diminution or absence of movement on respiration The classical signs of pleural fluid are usually present over the site of the empyema If the empyema is extensive it is common to find that the trachea and apex beat are displaced towards the affected side, on account of a shift in the position of the whole mediastinum In cases of empyema necessitatis, a tender soft swelling is found superficial to the ribs, and this usually has an impulse when the patient coughs (Fig 14)

Radiological appearances

A "latent" empyema shows as a dense opacity over which the ribs are approximated Periosteal new bone on the inner aspect of these ribs is frequently visible and is pathognomonic of chronic pleural disease, it is particularly marked when the infection is caused by actinomycosis If the empyema has been drained or has discharged spontaneously, or if a broncho-pleural fistula has developed, it is common to see a fluid level in the cavity, in which case it is usually possible to see the thickness of the fibrous tissue lining the cavity above the fluid level Skiagrams will also show the severity of the scoliosis, and the extent to which the diaphragm has been elevated and the mediastinum shifted as a result of the chronic fibrosis The films should be examined carefully for evidence of a foreign body within the empyema cavity, but, owing to the density of the opacity caused by the chronic empyema, it is often difficult to see a foreign body even when it is very radio-opaque, as, for example, with a rib sequestrum

Special investigation

If the empyema is not discharging externally, a sample of the fluid should be obtained by aspiration and sent for bacteriological investigation The operator may have difficulty in passing the needle through the chest wall, because of narrowing of the intercostal spaces, and he must be prepared to meet with considerable resistance to insertion of the needle, for the fibrous tissue lining the empyema is often tough The pus is usually thick, but it may be quite thin if the fluid has become sterile

When there is a sinus present or a tube *in situ*, a sample of the discharge should be collected in a test tube and examined bacteriologically It is particularly important to search for evidence of tuberculosis, because the treatment of a

tuberculous empyema may differ considerably from that adopted for a chronic pyogenic case With this in mind, the granulation tissue at the orifice of a sinus should be inspected and, if it is pale and oedematous, which suggests tuberculous infection, a small portion should be removed for histological examination

The length of an empyema cavity may be estimated in cases in which there is a sinus, by passing a fine gum-elastic bougie into it If the empyema is still being drained with a tube, the tube should be removed for a few moments and the patient rotated into various positions in order to see whether or not the drainage is adequate , the discharge of a considerable quantity of pus after removal of the tube is clear evidence that the drainage provided is inadequate In those cases in which there is reason to suspect the presence of intrinsic disease of the lung, such as carcinoma, appropriate investigation should be undertaken before treatment of the empyema is started

Treatment

A chronic empyema can be cured permanently only by obliteration of the cavity This may occur either as the result of re-expansion of the lung or in consequence of operative mobilization of the chest wall , in the latter case, the parietal pleura falls inwards to meet and fuse with the visceral layer where it covers the partially collapsed lung It is obvious that the functional and cosmetic result is much better when the cavity is obliterated by re-expansion of the lung , plastic operations on the chest wall should therefore be reserved for cases in which complete re-expansion of the lung proves to be impossible

Re-drainage

Many cases of chronic empyema will heal by re-expansion of the lung if proper dependent drainage is provided In those instances in which there is a discharging sinus or a drainage tube *in situ*, 5 millilitres of iodized oil should be introduced into the cavity through the sinus or tube, and skiagrams should be taken in the antero-posterior and lateral positions From these it is possible to decide the position which a tube should occupy in order to provide dependent drainage If a sinus leads into the bottom of the empyema, it may be dilated by the introduction of a laminaria tent, and a tube of adequate calibre subsequently fixed in position A sinus should never be dilated with bougies, for cases have been reported in which this procedure has been followed by the formation of a brain abscess If the sinus or tube does not lead into the bottom of the cavity, dependent drainage should be provided by the resection of a portion of rib, the site for this operation may be determined from the skiagrams taken after the introduction of iodized oil Resection of a rib gives an opportunity to obtain a biopsy of the cavity wall for examination for evidence of tuberculosis, and also *to inspect the interior of the cavity and thus exclude the presence of a foreign* body Therefore, even in those cases in which there is already an opening into the bottom of the cavity, resection of a portion of rib is usually preferable to dilatation of the sinus with a laminaria tent The drainage tube is cut flush with the chest wall and allowed to discharge into the dressings, because the mild negative pressure induced by water-seal drainage is insufficient to influence re-expansion of the lung in a chronic case It is possible to use much higher negative



FIG 17—Decort cat on for a chron c calc fied expectorated empyema Serial films showed that the calcium n the walls of the cav ty was breaking up and being shed nto the cav ty Th s case probably started 7 years previously as a spontaneous haemopneumothorax (b) 8 months after de cort cat on

(a)



(b)

mobilized to allow it to fall on to the visceral wall of the empyema It is held in this position by a pack placed on its outer surface The skin and superficial muscles are sutured over this pack, leaving a small opening for drainage The pack is removed after 10 days and the wound firmly strapped over a large pad of gauze and cotton wool With moderate and large-sized cavities this operation is preferable to Schede's operation, because it is more likely to be successful, and there is less deformity as well as a quicker convalescence

Complications of chronic empyema

Complications due to chronic toxaemia.—The most common complications are those which arise as the result of a chronic toxaemia, they include pulmonary osteoarthropathy, amyloid disease and nephritis The symptoms and joint effusions associated with pulmonary osteoarthropathy usually clear up quickly as soon as the chronic suppuration in the chest has been controlled It is important to realize that amyloid disease is also reversible, provided that it has not been present over a long period In many cases the symptoms and signs of amyloid degeneration improve greatly but do not disappear entirely, albuminuria is particularly apt to persist in a mild form Although nephritis is not common it is of particular importance to the surgeon, because patients with this complication are often bad risks for major surgical operations

Brain abscess.—A chronic empyema may also be responsible for pyaemic abscesses, particularly in the brain A persistent headache with nausea or vomiting should always lead the clinician to suspect a brain abscess, but, even if these suspicions are confirmed, all hope of the patient's recovery need not be abandoned, for numerous authenticated cases have been successfully treated during recent years by appropriate neurosurgery

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CHAPTER 3

HAEMOTHORAX

KENNETH M A PERRY

AETIOLOGY

Traumatic haemothorax

THE MAJORITY of cases of haemothorax or of haemopneumothorax follow trauma, which is usually associated with perforating wounds, but occasionally with non-perforating blows, even without fractured ribs This subject was well reviewed by Ogilvie (1950) who found haemothorax in 230 (58 per cent) of his 400 cases of chest injury Similar figures have been found by other authors Bradford and Elliott (1915) 156 (92 per cent) out of 170, Kay and Meade (1946) 455 (91 per cent) out of 500, and Thomas and Cleland (1945) 520 (70 per cent) in a series of 750 Traumatic pneumothorax becomes infected in about 22 per cent of cases, the infection being either introduced from without or resulting from inflammatory changes in the lung A large haemothorax is not so likely to become infected as a small one *Staphylococcus aureus* is the infecting organism in 50 per cent of cases, but other common invaders include streptococci, *Bacterium coli*, *Clostridium welchii* and other anaerobic organisms

Surgical trauma.—Haemothorax is a rare complication of artificial pneumothorax therapy, in which a vessel may be damaged by the needle, 7 cases were reported on by Miller and Rinkel (1947) Haemothorax may also result from thoracoscopy for adhesion section in about 4 per cent of cases, this is reviewed by Harold (1951)

"Spontaneous" haemothorax

Apart from these accidents there is a rare but definite entity of "spontaneous" haemothorax or haemopneumothorax In the majority of patients the disease occurs between the ages of 20 and 40 years, and in most of the cases described in the literature the condition has occurred in males When necropsies have been performed the cause has been found to be the rupture of a pleural adhesion which contained a blood vessel This accident may occur in a healthy individual or during the course of treatment for pulmonary tuberculosis Whenever the patient has died, haemorrhage has been the cause of death (Hopkins, 1937 Perry, 1938)

Haemothorax as a complication of other diseases

Rupture of either a syphilitic or a dissecting aneurysm of the aorta into the pleural cavity (East, 1939, Post, 1941) may occur as a terminal complication of these diseases

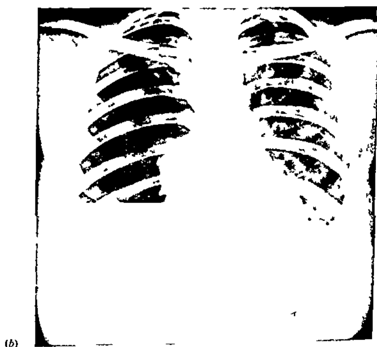
Endometrioma of the pleura is a rare condition, which occurs in women and will produce spontaneous haemothorax, it is usually associated with endometrioma elsewhere so that ascites may also be present (Nicholson, 1951) (Fig 18)

Apart from these causes of frank haemothorax, blood-stained pleural effusion



(a)

FIG 18 —(a) Skiagram of a woman aged 39 years with a large haemothorax and ascites resulting from endometriomas of ovaries colon and pleura (b) after removal of the blood stained effusion



(b)



Fig 19 —Sk agram of a mun who developed an inter nal haemorrhage nto h s left pleural cav ty three weeks after fracturing 3 r bs S x p nts of blood were re moved at thorac otomy

occurs as a complication of carcinoma of the lung pulmonary tuberculosis Hodgkin s disease pneumonia cirrhosis of the liver reticulosis scarlet fever smallpox arterial hypertension and mitral stenosis (Vaizey and Perry 1940)

SYMPTOMATOLOGY

In cases which do not result from trauma the patient usually gives a history of sudden onset of sharp stabbing pain in the side of the chest although sometimes it is located in the upper part of the abdomen This is followed by shortness of breath If the bleeding is extensive the patient will be shocked and collapsed He will rapidly become pale On examination he may be febrile There will be a raised pulse and respiration rate If the haemothorax is rapid he will give the appearance of a patient suffering from internal haemorrhage but if it is less severe and if the lung is torn at the time that the haemorrhage occurs he will have the appearance of a patient with a pleural effusion or a pneumothorax The trachea is likely to be displaced away from the side of the lesion On the side involved there will be diminished movement an absence of tactile vocal fremitus impaired percussion notes or stony dullness and distant or absent breath sounds If there is a haemopneumothorax there will be hyper resonance above the dullness Sometimes owing to irritation of the diaphragmatic pleura by the blood there may be rigidity of the upper abdomen This may be very marked and simulate the rigidity of a perforated peptic ulcer Of the cases of

this disease reported in the literature more than half have been operated on with this mistaken diagnosis

THE FATE OF BLOOD IN THE PLEURAL CAVITY

The fate of blood in the pleural space has long been a debated subject, but as long ago as 1834 LeBlanc and Trousseau showed that blood injected into the pleural cavity of horses first clotted and then was absorbed, and in 1916 Denny and Minot showed that blood stained fluid in the pleural cavity consisted only of serum and cells

The problem has recently been studied by Ogilvie (1950) and by Harold (1951) and it is now generally accepted that blood in the pleural cavity clots at once, but that this clot is soon broken up and dispersed by the movements of the heart and lungs The blood is thus defibrinated and the fibrin either is deposited on the pleura or sinks in clots to the bottom of the pleural space The pleural cavity thus becomes like a blood-cyst lined with fibrin, thicker on the parietal than on the visceral pleura The blood which would then be aspirated resembles whole blood but, in fact, cannot clot since it has lost its fibrinogen This blood acts as a foreign body and stimulates the outpouring of fluid, which reaches a maximum within 3 days The blood is thus diluted and the fibrinogen concentration rises, thus making secondary clotting possible in the second week, this can be prevented by early aspiration Sellors (1945) gives evidence supporting this theory, by showing that there is a steady fall in haemoglobin concentration in the fluid, and as steady a rise in fibrinogen over the period of 7 days after the haemorrhage If the blood is not removed an organizing haemothorax results This process has been described by Samson and Burford (1947) Usually the pleura remains normal though coated with fibrin, but in some cases the scar tissue extends through the pleura into the interstitial tissue of the lung and this may develop more rapidly if an empyema is present Ogilvie (1950), however, quotes cases in which dense fibrous adhesions have been found at operation in the second week, even in cases of sterile organizing haemothorax

PROGNOSIS AND TREATMENT

Ogilvie (1950) showed that the average duration of illness, in cases of sterile traumatic haemothorax, adequately treated by aspiration was about 6 weeks, whereas if the treatment was inadequate this time was doubled In his whole series of 230 cases, which included infected, clotted and organizing haemothoraces operation was necessary in 63 cases (27 per cent) and that in these complicated cases the duration of illness was much longer and physical and psychological results were less satisfactory, although there was only 1 death

In cases which occur during artificial pneumothorax therapy treatment, if adequate, is so satisfactory that a useful surviving artificial pneumothorax is the rule Deaths are rare, as is also the case in spontaneous haemothorax

Endometrioma may be excised and an artificial menopause induced by radiation therapy In these cases also the prognosis is good The prognosis of blood stained effusions depends on the aetiology of the underlying disease

Medical treatment

If all the fluid can be removed from the chest and the bleeding stops, aspiration and treatment of the anaemia may be the only treatment required Aspirations should always be started as soon as possible They should be continued so that the pleural cavity is kept dry Intrapleural injection of streptokinase will help to break down fibrin If the anaemia is not severe, the patient responds quickly to iron, but in some instances blood transfusion will be necessary

Surgical treatment

In cases in which blood clot is present, aspiration will not be possible, these cases have been described as clotted haemothorax Early thoracotomy with removal of the blood clot is then essential Blood clot left in the pleural cavity causes rapid thickening of the pleura and frequently calcification

Haemorrhage may be so severe as to cause the death of the patient In such cases immediate thoracotomy, with removal of the blood clot and tying of the bleeding point, is a life saving operation

When the blood clot becomes infected or an empyema develops, chemotherapy, systemic and intrapleural, with the drug to which the infecting organism is sensitive is indicated There is much to be said however, for giving penicillin in all cases as a prophylactic If an empyema develops it may require drainage

In cases of clotted or organizing haemothorax, decortication may become necessary, if surgery is needed on these lines it is probably best carried out in the fourth week of the illness

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If all the fluid can be removed from the chest and the bleeding stops, aspiration and treatment of the anaemia may be the only treatment required Aspirations should always be started as soon as possible They should be continued so that the pleural cavity is kept dry Intrapleural injection of streptokinase will help to break down fibrin If the anaemia is not severe, the patient responds quickly to iron, but in some instances blood transfusion will be necessary

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CHAPTER 4

SPONTANEOUS PNEUMOTHORAX

KENNETH M A PERRY

AETIOLOGY

IN PNEUMOTHORAX the air collects between the layers of the pleura causing it to become a real instead of a potential space When fluid, pus or blood are also present it is known as a hydropneumothorax, pyopneumothorax, or haemopneumothorax Air may gain access to the pleural space through the visceral pleura, from the lungs, through the chest wall as a result of penetrating wounds, from the mediastinal organs, through the diaphragm from some hollow viscus, or from gas-producing organisms infecting the pleura

Aetiologically the causes of spontaneous pneumothorax may be divided into three groups The first group includes symptomatic pneumothorax which may result from rupture of the parietal pleura as a result of trauma caused by penetrating wounds, when a sucking pneumothorax may occur, or non penetrating injuries which result in laceration of the pleura with or without fractured ribs The history of injury will be evident The visceral pleura may be perforated in empyema and also from lung abscess, particularly staphylococcal lung abscesses in children, which may result from bronchogenic infection or staphylococcal septicaemia (Collis and Foster Carter, 1940, Butler and Perry, 1940)

The pleura may also be perforated by carcinoma of the lung or punctured during operations of needling of the chest for paracentesis Bilateral pneumothorax is a post operative cause of death in thyroidectomy and tracheotomy and the recognition of the condition may be a life-saving measure (Keis, 1934, Barrie, 1940, Ackerman and Bucker, 1941) Oesophageal cancer or accidental perforation of the oesophagus during the passage of an oesophageal bougie or oesophagoscope may give rise to spontaneous pneumothorax It sometimes complicates sub-phrenic abscess or carcinoma of the stomach and colon and it can result from incision of the parietal pleura during operations on the kidney

The second group are those cases that develop a spontaneous pneumothorax as a result of perforation of the lung by manifest active pulmonary tuberculosis or as a complication of artificial pneumothorax therapy Sita-Lumsden (1949) recorded its occurrence as a complication of artificial pneumoperitoneum therapy, the air tracking to the pleural cavity through a persistent pleuroperitoneal canal A pneumothorax may, of course, be induced accidentally when the lower intercostal route is employed for the induction or maintenance of pneumoperitoneum

The third group is where it occurs in the apparently healthy The condition is then known as benign spontaneous pneumothorax It is most frequent between the ages of 15 and 35 years It is eight times more common in males than in females

Rupture of the lung, with resultant pneumothorax, may occur in the

newborn following a normal birth (Ellison and Carabelli 1940) It used to be thought that benign spontaneous pneumothorax was the result of a sub-pleural tubercle rupturing into the pleura and it is possible that in rare instances this is the cause of the condition It is quite evident however that if this occurred the pleura would be infected with tubercle bacilli and would immediately give an acute febrile reaction with the outpouring of fluid and raising of the sedimentation rate a reaction which never occurs in benign spontaneous pneumothorax Kjaergaard (1932) and Perry (1939) followed groups of cases and showed that it was rare for them subsequently to develop pulmonary tuberculosis which is in marked contrast to patients with pleural effusion There is no doubt



Fig 20 —Microphotograph of section of lung showing an intact emphysematous bulla with a ruptured one beside it This caused a spontaneous pneumothorax

that a vast majority of the cases result from the rupture of sub-pleural air vesicles Such an air vesicle may be a result of compensatory emphysema from chronic pulmonary tuberculosis where the disease is quiescent It is an infrequent complication of the focal emphysema of pneumoconiosis The vesicles are sometimes of congenital origin but usually result from non tuberculous scar tissue formation (Fig 20) or chronic hypertrophic emphysema The vesicles may be readily seen through a thoracoscope and have been described by Brock (1948) He found that generalized emphysema and localized bullous emphysema were the commonest causes next to these small apical bullae apical scars or tearing of the lung Some cases resulted from either localized cystic lung or generalized polycystic disease and a few cases appeared to arise from areas on the surface of the lung resembling cuckoo spit Spontaneous pneumothorax may be

complicated by mediastinal emphysema, which may spread to involve the pericardium and give rise to a pneumopericardium The air may also spread to cause surgical emphysema of the head and neck and even the arm and chest wall

CLINICAL SYMPTOMS

The patient most often complains of pain in the chest on the side affected The pain is usually of sudden onset so that the patient can record the exact moment and what he was doing at the time, rarely, the onset may be gradual with the patient unable to date it at all The pain is generally in the side of the chest but sometimes it is situated in the shoulder or the back, and rarely, the patient complains of pain in the upper abdomen When the pain is abdominal it may easily be mistaken for a perforated peptic ulcer It most often occurs early in the morning on getting out of bed, but it sometimes occurs later in the day, or after a prolonged rest such as a visit to the cinema The pain is followed by shortness of breath, especially on exertion, and sometimes the patient may be cyanosed Among the less common initial symptoms haemoptysis is perhaps the most frequent, a little blood stained mucus being coughed up, probably arising from the lung at the point of rupture Vomiting may occasionally occur as an early symptom In a few cases the patient is greatly shocked as a result of the disease

Physical signs will depend to a great extent on the amount of air in the pleural cavity There is displacement of the trachea and apical beat away from the affected side of the chest, diminished movement and diminished tactile vocal fremitus, hyper-resonance, diminished or distant breath sounds, and absent voice sounds, and in some cases the coin sound elicited by placing a coin on the chest and tapping with another The heart and trachea are not likely to be greatly displaced but all degrees of displacement may occur In a left sided pneumothorax the trachea may lie under the right sternomastoid muscle and the apical beat be found on the right of the sternum When this occurs the patient is likely to be cyanosed and orthopnoeic In most cases the patient is afebrile but in those cases of tension pneumothorax which result from a valve like action of the pleural perforation there may be a slight rise of temperature and pulse rate, both of which are immediately relieved by removal of air

The diagnosis can be established conclusively by x ray examination which will show air in the pleural cavity with usually no disease in the underlying lung There is often congestion of the opposite lung from increased blood supply, which is always present and should not be mistaken for tuberculous infiltration Christie and McIntosh (1936) showed that immediately the lung collapses the [illegible] As a result [illegible] the blood [illegible] lways seen in the radiographs, and also the rareness of cyanosis in this disease If there is a tension pneumothorax a mediastinal herniation of pleura to the other side may occur and show itself as a semi circle in the other lung field with the hilum as the centre Such a shadow is not infrequent in the course of artificial pneumothorax therapy The sedimentation rate is always normal and in most cases there is no sputum for examination

DIAGNOSIS

The history of the pain is sometimes misleading and the case arriving at hospital complaining of pain in the side or back is easily labelled rheumatism, lumbago, or fibrositis When the pain is in the chest it may be labelled pleurisy, if, however, the condition is considered and skiagrams are taken, the diagnosis should not be missed In cases where there is displacement of the mediastinum the diagnosis is easy because in these cases other signs are well marked It may

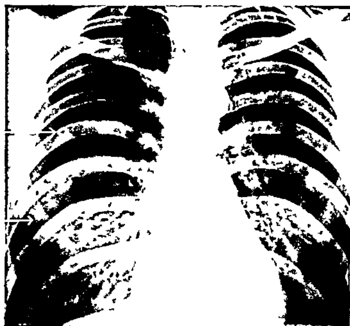


Fig 21 —Skiagram of a young man showing a single benign spontaneous pneumothorax of medium extent (The arrows indicate the lung periphery)

however be difficult to differentiate, even radiologically a case of spontaneous pneumothorax from one with a very large emphysematous bulla Congenital bronchial cysts may grow to a size that will give physical signs and radiological appearances superficially resembling a spontaneous pneumothorax Such cysts are frequent in infants A large sub-phrenic abscess containing gas is also liable to be confused with a spontaneous pneumothorax In cases of bilateral spontaneous pneumothorax it is not possible to compare the signs on the two sides but the patient is usually seriously ill and there is thus no possibility of the condition being missed, skiagrams will establish the diagnosis In relapsing or alternating pneumothorax the diagnosis is not so difficult as in the first attack, and often the patient makes the diagnosis himself as soon as the symptoms reappear

PROGNOSIS

The course of benign spontaneous *pneumothorax is favourable in the majority* of cases, few necropsies being recorded in the literature The condition may however become recurrent or chronic, and 71 such cases were described by Brock in 1948 In most cases the lung expands in a period of six weeks, but in a few cases the pneumothorax may persist for months or years Recurrence occurs in about 15 per cent of cases *When recurrence does occur* the patient may have five, six, or even more attacks, and these may be on one or both sides

TREATMENT

Puncture wounds of the chest caused by a knife or sharp instrument seal quickly and a closed pneumothorax is then established As a result, however, of trauma from jagged objects or gunshot wounds, a sucking pneumothorax may develop and become established under atmospheric pressure This impedes the action of the heart and lungs, and it is most important to close the wound in the chest wall as soon as possible *A thick gauze pad may be fixed over the opening* as first aid treatment But on reaching hospital the wound should be excised and sutured as soon as possible

The principles of treatment of closed pneumothorax are the same whether the lesion occurs in the apparently healthy, results from trauma or from any other cause *In milder cases the patient frequently does not seek advice immediately* and when he does no treatment is required In more severe cases rest in bed for one week should be sufficient to allow the perforation to heal and the air to start absorbing Sanatorium treatment is unnecessary except in tuberculous patients In the benign cases the patient should be warned that there is a certain risk of recurrence of the condition, this being greatest during the first year Tension pneumothorax usually develops slowly although sometimes as an exacerbation two or three days after the onset of the original pneumothorax The patient is greatly distressed, orthopnoeic, and cyanosed, but the condition may be immediately relieved by the insertion of a Foster-Carter needle attached to an under water drain Pneumothorax occurring in cases of severe generalized emphysema or where bilateral pneumothoraces have occurred may also require the insertion of the Foster-Carter needle Such patients should also receive oxygen therapy, either by means of bilateral nasal catheters, a nasal mask or oxygen tent *The most satisfactory method of treating troublesome chronic* or relapsing and recurring pneumothorax is by means of *pleurodesis by silver* nitrate, 5–10 minims of 10 per cent solution is injected into the pleural cavity A febrile reaction follows which usually lasts about five days and is accompanied by effusion of varying size When it has subsided it is advisable to show that the pleural space is obliterated by demonstrating that it is not possible to *induce an* artificial pneumothorax

In cases where a lobe is grossly abnormal, usually from congenital cystic formation excisional surgery may be the best form of therapy

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CHAPTER 5

CALCIFICATION OF THE PLEURA

I C LODGE PATCH and KENNETH M A PERRY

INCIDENCE

SINCE the days of Aristotle pathological calcification of the pleura has been recognized, and *in the 18th century Morgagni (1682–1771)* drew attention to the condition Since radiological examination of the chest has become common it has been realized that the condition is not infrequent If the incidence is estimated by radiography alone it will be found in unselected cases to be less than 0 2 per cent

In a series of 1,845 cases Ulrich (1930) found a percentage of 0 87, Pritchard (1923) in 7,000 cases found 0 12 per cent, in Taylor and Schwartz's (1937) 10 000 cases there was 0 1 per cent, and 0 18 per cent were found in 15,000 cases by Floyd and Hepburn (1939) In contrast, however, Behrendt (1930), in reviewing ex-servicemen found an incidence of 1 2 per cent in 886 cases Even allowing for the greater risk of injury to men especially in wartime they show pleural calcification much more commonly than women Thus Head (1934) found it in 47 men compared with 8 women and Odessky and Klioner (1932) in 11 men and 2 women

AETIOLOGY

Most cases result either from trauma, where there has been haemorrhage into the pleura cavity, or from inflammation of the pleura which may have resulted either in a serous pleurisy or an empyema (Fig 24) It occurs in some cases of spontaneous pneumothorax, probably as a result of bleeding into the pleural space (Fig 22)

TABLE

CAUSE OF CALCIFICATION IN 70 CASES (HEAD 1934)

Cause	Number of cases	Percentage
Gunshot wounds	17	24 3
Active tuberculosis	8	11 4
Inactive tuberculosis	3	4 3
Pleural effusion	12	17 1
Dry pleurisy	7	10 0
Respiratory disease—		
influenza	1	
pneumonia	1	14 3
pneumonia and empyema	3	
chronic lung disease	4	
empyema	1	
No respiratory disease	13	18·6

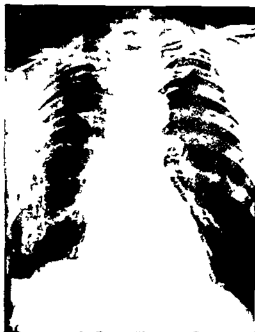


Fig 22 —A man aged 50 years w th a r ght chron c spontaneous pneumo thorax and calcified pleura

S a n (1933) ... 'Vest (1906) occurs as ... nd Tonkin ... d Church and Ellis (1950) mention cystic changes but there is no recorded case of calcification arising in this condition

Head (1934) collected 70 cases of pleural calcification from the literature He lists the causes as shown in the Table

PATHOLOGY

Calcification is commonly unilateral and is related to the aetiological cause it is only bilateral in the idiopathic cases The calcium is deposited between the layers of the pleura and while it is often loosely attached to the pleural surface it is in fact intrapleural (Stott and Cotton-Cornwall (1933) Head (1933) Goulhoud (1906) Boswell and Dredge (1940)) In extensive calcification it may appear as a crust over the pleura (Hill 1933) The mechanism by which the calcium is deposited is uncertain Head and Boswell and Dredge (1940) consider that the pleura acts as a dialysing membrane and the calcium is deposited as a superficial crust as the last of the fluid is absorbed But Pritchard (1923) and Hsu and Wang (1936) suggest that the fluid or blood is encapsulated by thickened pleura and slowly absorbed The nutrition of the pleura is impaired and hyaline de generation ensues in this degenerate tissue so that calcium is then deposited



Fig 23 — Idiopathic calcification of the pleura in a man aged 50 years who had an abscess pointing on the surface

Fig 24 —Calcified pleura resulting from a pleural effusion in childhood

While Pritchard (1923) and Head (1933) did not find local or remote evidence of tuberculosis, Taylor and Schwartz (1937) regarded calcification of the pleura as analogous to the calcification of tuberculosis in any other organ Haematogenous tuberculosis produces a caseous pleuritis, with or without effusion and subsequent calcification In three necropsies they found the association of calcification and caseation in the pleural layer Haematogenous tuberculosis was found elsewhere in all three subjects It is however quite clear that tuberculosis cannot be the cause in all cases since calcification is found in non tuberculous patients

The size of the calcium deposits may vary from small spots (Hill 1933) to single plaques (Pritchard, 1923) and multiple plaques (Pallasse and Martinon 1937) and extensive shells (Hill 1933, and West 1906)

Empyema is commonly found in association with calcification of the pleura (Pritchard, 1923) and Taylor and Schwartz (1937) regard this as a common complication when the pleural layers are not adherent It is probable that the plaques erode the lung tissue and infection passes from a bronchus Where the pleura is adherent small abscesses may develop which may point on the surface

CLINICAL FEATURES

There are usually no symptoms in association with the condition but if it is extensive, shortness of breath may develop and cough is sometimes associated If the plaques give rise to pus formation there may be fever and an abscess may appear in the chest wall (Fig 23) On examination the chest is often fixed and flattened and shows an impaired percussion note and diminished air entry (Beverley 1937 1942, Hsu and Wang 1936 Passalacqua and Farias 1939 Stott and Cotton Cornwall 1933) There may be scoliosis (Hill 1933)

TREATMENT

In the absence of symptoms no treatment is indicated If the calcium plaque or *sheet is loose or causing symptoms it may be removed (Head 1933 and Goul*houd, 1906) If there is marked shortness of breath and the chest wall is fixed decortication of the lung may be advisable

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CHAPTER 6

TRANSIENT PULMONARY INFILTRATION WITH EOSINOPHILIA

JAMES L LIVINGSTONE

DEFINITION

TRANSIENT pulmonary infiltration with eosinophilia (Löffler's syndrome) is defined as a group of clinical conditions, varying in duration and severity, in which transitory pulmonary opacities and eosinophilia in the peripheral blood are cardinal features the histological picture suggests an antigen antibody tissue reaction

HISTORY

In 1932, Löffler described 5 cases and in 1936 a series of 51 cases with minimal symptoms, transitory pulmonary infiltrations and eosinophilia up to 22 per cent without leucocytosis Subsequently, Loffler under Maier (1943) found ascaris infestation in a proportion of cases, particularly in soldiers of the Swiss army Von Meyenberg (1942) reported post mortem examinations of 4 soldiers in whom broncho-pneumonic patches were found with infiltration of eosinophilic cells Two of these patients had ascaris worms in the intestine

Since then numerous papers have appeared from all over the world describing groups of cases showing eosinophilia and transient pulmonary infiltrations these fall into 4 groups (1) Infestation with parasites, for example *Ascaris*, *Necator americanus* (2) Toxic reactions from drugs, from serum injections, and sensitivity to foods and plants (3) Bacterial infections, particularly in patients with asthma or other allergic conditions, in rheumatic fever and brucellosis (4) Tropical eosinophilia (pulmonary eosinophiliosis) occurring chiefly in India and Ceylon In 1940, Frimodt Moller and Barton reported 175 cases and in 1943 Weingarten reported 81 cases and found that arsenic was specific therapy

Rich and Gregory (1943) produced polyarteritis nodosa in rabbits by sensitization to horse serum and they found a specific histological picture of fibrinoid necrosis of connective tissue occurring as a result of this antigen antibody reaction Harkavy (1943) and Bergstrand (1946) in post-mortem material in asthmatic patients reported arterial lesions, fibrinoid necrosis and rheumatic granulomas essentially similar to the lesions in rabbits produced experimentally by Rich and Gregory

AETIOLOGY

The possible causes of this condition may be divided into 4 groups (1) Infection with parasites, for example *Ascaris lumbricoides N americanus, Ankylostoma braziliense, Fasciola hepatica* and *Trichinella spiralis* Acari (mites), *Entamoeba histolytica* and microfilaria have been suggested as possible causes (2) Toxic or allergic reactions to (a) drugs such as the sulphonamides, organic arsenicals, and thiouracil, (b) injections of foreign sera, (c) foods such as egg and tomato,

and (d) pollens or certain plants such as privet (*Lingustrum*) (3) Bacterial infections of the respiratory tract, particularly in patients with an allergic diathesis (such as asthma, hay fever, and eczema) Acute rheumatism and brucella infections have been reported (4) Tropical eosinophilia (pulmonary eosinophiliosis) This appears to be a clinical entity, in that although the cause is unknown, arsenic is a specific remedy

MORBID ANATOMY

In 1942, von Meyenberg reported post-mortem findings in 4 soldiers, 3 of whom died suddenly as a result of accidents and 1 from tetanus He described consolidations in the lungs without fibrin formation, with eosinophilic infiltration, giant cells in the alveoli and thrombophlebitis and rheumatic granulomas in the interstitial tissue Eosinophilic infiltrations were present in the liver and elsewhere Two of these men had ascaris worms in the intestine Harkavy (1943) in post-mortem examination of four asthmatics found eosinophilic infiltration of the inter-alveolar septa with arterial lesions, varying from simple intimal thickening up to acute necrosis of the arterial wall with thrombosis Bergstrand (1946) described the post-mortem findings in four asthmatics with infection of the respiratory tract, transient lung infiltrations and eosinophilia in the blood Besides polyarteritis nodosa lesions in various stages, he found rheumatic granulomas, as described by Aschoff, in the interstitial tissues of the lungs; there was oedema and swelling of the collagen fibres which split up into fine fibrils, with large fibroblasts which sometimes form multinucleated giant cells eosinophil cells were frequent, and considerable formation of fibrous tissue was present Necrosis in the centre of the granulomas was frequently found, and conglomerate granulomas were seen in the interlobar septa which were visible to the naked eye as greyish white streaks in the lungs These appearances are essentially identical with those seen in rheumatic pneumonia and in subcutaneous rheumatic nodules

CLINICAL FEATURES

The clinical features of this condition may be considered in 3 groups according to the aetiology

Helminth and parasitic infestations, serum and drug reactions

Symptoms may be entirely absent or minimal with lassitude, cough, chest pains, slight fever and occasionally sputum, which is rarely blood stained Physical signs are either absent or confined to a patch of fine râles

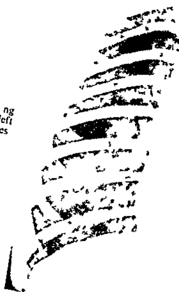
Skiagrams of the chest show varying types of nodular opacities which disappear in 10–21 days

The blood shows a moderate leucocytosis with an eosinophilia usually below 20 per cent

A search for parasites or ova should be made and treatment obviously depends upon the cause

Bacterial infection, especially of the respiratory tract

In this group there is a history of previous allergic conditions (asthma, eczema,





25 — Resolv ng pneumonia in left ı d and lower zones





Fig 26 Further resolut on at left base but fresh fa nt round opacity in r ght fourth space



FIG 27 — Extensive opacity in right mid and *lower zones shown on* the lateral view to be in the anterior segment of the upper lobe and mid basic segment of the lower lobe Patient had *been free of symptoms* for three days

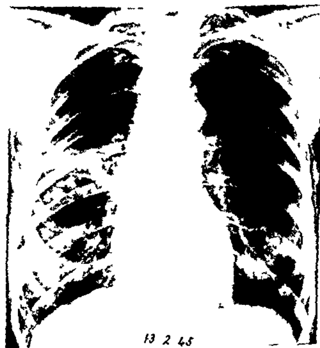


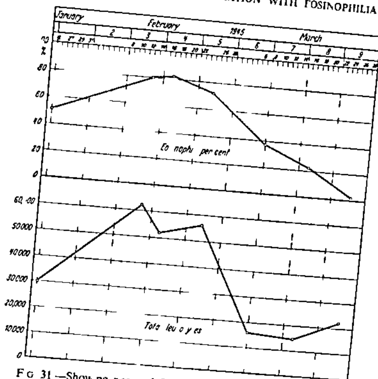
FIG 28 — Some clearing of the opacity



Fig 29 — Cons derable ncrea e n the card ac shadow w h pleural reac on at bo h bases



F c 30 S ll gross card ac enla gement w th clearing of the pleurisy at the bases



F G 31 —Show ng r se and fall in eos noph ls and fluctuat on of leucocyte count of Case F E

Case Report F E

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gns suggested

March 15 1945 Temp 100[illegible] pulse 100 Anasarca ascites Liver enlarged Sputum thick [illegible]

[illegible] d

[illegible] t

per cent

March 26 1945 Improving good diuresis with Neptal

March 30 1945 Patient discharged herself from hospital She remained bed ridden in cardiac failure

July 1945 Died No necropsy allowed

Investigations

Sputum few organisms No tubercle bacilli many Charcot Leyden crystals and eosinophils Pus cells were numerous

Stools no ova or parasites

Casoni test negative

[illegible] ormality

[illegible] 00 c mm

urticaria and rhinitis) in more than half of the cases, and a family history of allergy is common The clinical picture is usually that of recurrent attacks of bronchitis with severe asthma Pleural effusions are common and may be bilateral, with many eosinophil cells in the fluid , the proportion of these cells may differ markedly in the fluid from one side as compared with the other The heart may dilate, and resume its normal size at a later date The spleen and liver are often enlarged, the lymph nodes less frequently Nodules may appear in the subcutaneous tissues, muscles or tendon sheaths, similar to large " rheumatic nodules " in rheumatic fever Albuminuria with red cells and casts may occur periodically or persistently, and hypertension may supervene with terminal cardiac or renal failure

There is a leucocytosis from 20,000 to 60,000 per cubic millimetre, with an eosinophilia up to 84 per cent during the acute phases, falling to normal between attacks

Radiographic examination shows pulmonary opacities, rather large and asymmetrical, which persist for days or weeks and then disappear completely These opacities do not appear to be areas of segmental collapse, and come and go without definite relation to the acute attacks of infection (Figs 25–30)

The majority of recorded cases have died following an illness of 3–15 months after the onset of the eosinophilia A number of patients are still alive however, and apparently recovering after observation for 2–4 years It is obvious that this group, if not identical with polyarteritis nodosa is very closely related to that condition

There is no specific treatment Antihistamine drugs may be tried both during the attack and prophylactically

Tropical eosinophilia (pulmonary eosinophilosis)

This condition appears to be a clinical entity, although the aetiology is unknown It occurs chiefly in India and Ceylon, although sporadic cases have been reported in America, North Africa, China, Australia, the Pacific Islands, Egypt and elsewhere

Viswanathan (1948) reviews the subject and reports the post mortem findings in one case in which there were nodules in the lung showing eosinophilic infiltration and a similar picture to that described by Bergstrand

Clinical features

Acute type —The onset is sudden with fever from 102° to 104° F with cough and rapid respirations simulating acute bronchiolitis The signs are those of acute bronchitis and occasionally of patchy broncho pneumonia Radiographic examination shows a patchy mottling confined to one part of the lung, or uniformly distributed in all fields There is a leucocytosis ranging from 12 000 to 80,000 per cubic millimetre with from 20 to 80 per cent of eosinophils

If untreated, about a quarter of the cases clear up spontaneously in from 3 to 6 weeks, but the remainder become chronic cases

Chronic type —The onset is insidious with malaise, lassitude, anorexia and low grade fever from 99° to 101° F Cough may be noticed at the onset or more commonly after a week and is unproductive, paroxysmal and often most distressing at night Small blobs of viscid sputum are expectorated with difficulty and there is marked dyspnoea on coughing or on exertion, rather than typical nocturnal asthma The spleen is palpable in about half of the cases, and occasionally there is hepatomegaly and enlarged lymph glands Radiological opacities are seen during the earlier stages of the disease, and occasionally persist for months, coarse mottling may be present throughout the lungs or confined to one part cavitation may be seen but is uncommon In the later stages, heavy hilar shadows may be the only abnormality

The erythrocyte sedimentation rate is usually raised Viswanathan and [illegible]

[illegible]

syphilis The reaction becomes negative after treatment with arsenic

Weingarten (1943) reported 81 cases and observed, when treating one patient suffering from syphilis with neoarsphenamine, that there was a rapid improvement in the symptoms and in the eosinophilia He then discovered that arsenic was a specific cure

The usual course of neoarsphenamine is 0 15 gramme followed by 7 weekly injections of 0 3 gramme After the first dose there is usually an exacerbation of symptoms with an increase in the eosinophilia The clinical symptoms and signs quickly disappear but the blood does not return to normal for several weeks after the completion of treatment

In real asthma or in helminth infestation, organic arsenic does not improve the condition nor is there the preliminary increase in eosinophilia which are points in differential diagnosis

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CHAPTER 7

POLYARTERITIS NODOSA (PERIARTERITIS NODOSA)

JAMES L LIVINGSTONE

DEFINITION

POLYARTERITIS nodosa is a syndrome of unknown aetiology, marked by inflammatory reactions of the collagen connective tissue, particularly in the walls of arteries, and associated with a prolonged febrile course and multiple lesions throughout the body and commonly ending fatally

HISTORY

Kussmaul and Maier in 1866 gave the original description of the condition and correlated the clinical symptoms with the lesions found at autopsy Since then details of some 400 further cases have been published, particularly during the past 15 years although the majority have been discovered at autopsy, an increasing number have been diagnosed in life on clinical grounds and confirmed by biopsy of nodules Page (1947) records a case in which peritoneoscopy confirmed the clinical diagnosis, the nodules being seen on the arteries of the stomach and colon During recent years an increasing number of proved cases with recovery have been recorded

MORBID ANATOMY

The essential lesion is an acute fibrinoid necrosis of the collagen tissue, especially in the walls of the smaller arteries and arterioles There is oedema round the necrosed collagen, with infiltration of endothelioid cells and lymphocytes, and eosinophils are frequently seen in large numbers At a later stage the lesion is identical with an Aschoff's node, still later, fibroblastic proliferation occurs, and when healing is complete a small fibrous scar remains The arterial lesions start in the media, but there is always a proliferation of the intima at the site, and intravascular thrombosis, rupture of the arterial wall and aneurysm formation are all common Lesions in different stages of development may be seen at the same time, and have been found in practically every organ and tissue of the body The kidneys, liver, lungs, spleen, heart, small intestine, pancreas, suprarenal glands and uterus are most frequently affected, and it is relatively uncommon to find nodules in the superficial arteries which are available for clinical diagnosis

In the kidney the afferent arterioles to the glomeruli are frequently involved, causing hyalinization of the glomeruli, indeed, changes identical with those found in malignant hypertension may be found, and many patients with polyarteritis nodosa die from renal failure with hypertension and uraemia

In biopsy specimens particularly, it may be difficult to detect the typical arterial involvement unless serial sections are cut, and reticulum stains should be used The degree of eosinophilic infiltration is very variable (Figs 32–35)



Fig 32 — Acute polyarteritis nodosa (low power) Artery in striated muscle Fibrinoid necrosis and cellular infiltration involving a segment of the vessel wall Haematoxylin and eosin × 105 (reduced by ½ on reproduction) (*By courtesy of Professor H A Magnus*)



Fig 33 — High power of Fig 32 showing acute fibrinoid necrosis of media with cellular infiltration of adventitia and periarterial tissues Haematoxylin and eosin × 370 (reduced by ½ on reproduction) (*By courtesy of Professor H A Magnus*)

History Female aged 41 years Six months history of loss of weight and swelling of ankles Admitted to hospital with irregular pyrexia to 102 with purpura acne rosacea and splenomegaly Blood pressure 140 105 Haemoglobin 56 per cent with a normal leucocyte count She developed pain in the chest from a pulmonary infarct and died in coma after sixteen days (Figs 32 33)

AETIOLOGY

The cause of polyarteritis nodosa is as yet quite unknown Theories postulating a specific bacterium, virus or chemical toxin have been without scientific proof

The specific histological picture of an acute reaction in the collagen connective tissue is in many ways similar to the histology in acute rheumatism and is also found in lupus erythematosus disseminatus dermatomyositis, scleroderma and certain chronic types of transient pulmonary infiltrations with eosinophilia The



FIG 34 — Healed stage of polyarteritis nodosa in small renal artery (low power), showing intimal thickening and segmental fibrosis with aneurysmal bulging of media Picro-Mallory, × 70 (reduced by [illegible] on reproduction) (*By courtesy of Professor H A. Magnus*)

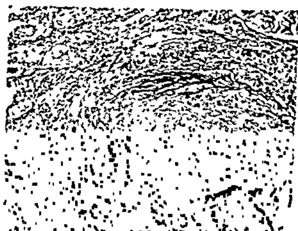


FIG 35 — High power of Fig 34 Picro-Mallory, × 140 (reduced by [illegible] on reproduction) (*By courtesy of Professor H A Magnus*)

History
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(*Figs* 34,

theory of an allergic antigen-antibody reaction has been postulated but until recent years there was no known method of producing similar lesions in animals Rich (1942), however, noted a marked increase in the incidence of polyarteritis nodosa in the post mortem department of the Johns Hopkins Hospital from 1935, when sulphonamides were introduced, as compared to the previous 25 years He found that a high proportion of these patients had suffered from serum sickness (from Felton's anti-pneumococcal serum) or from sulphonamide drug reactions in life

He then sensitized rabbits by giving them a very large dose of horse serum intravenously (corresponding to 200–300 millilitres in humans) and noted that approximately 75 per cent developed serum sickness 7–10 days later, as evidenced by pyrexia and flushing of the ears. By the fourteenth day all the rabbits were sensitized to horse serum as shown by the dermal skin tests. He then gave these rabbits varying doses of horse serum intravenously, and found that while some animals died immediately of anaphylactic shock, a number survived. These were killed at varying times and showed typical polyarteritis nodosa in different stages of development. The heart muscle and valves showed reactions identical with acute rheumatic myocarditis and endocarditis in man. Rich's work has been substantiated by others.

The inference is that at any rate some cases of polyarteritis nodosa are caused by an antigen-antibody reaction. This does not explain however why polyarteritis nodosa is much more common in males and why it occurs in persons who have never shown any clinical evidence of allergy or toxic reactions to drugs or sera. As a working hypothesis, however, Rich's investigations are of considerable significance.

CLINICAL MANIFESTATIONS

General

Males are affected at least twice as often as females. Although a case has been reported in an infant, the common age is from 25 to 55 years. The onset may be abrupt or insidious, with muscular aches and pains, irregular pyrexia and tachycardia, with progressive anorexia, loss of weight, and weakness as the most prominent symptoms. Muscular tenderness and splenomegaly are common. Tender nodules may be found in the subcutaneous tissues or muscles, and occasionally a tender thickening is felt in a superficial artery, such as the temporal artery, which of course suggests the diagnosis. The retinal vessels may show localized areas of thickening, but more frequently exudates and retinal detachments are seen.

Remissions of some weeks' duration may occur in the course of the disease, followed by relapses with involvement of other areas.

Pathological investigations commonly show a moderate secondary anaemia, with a polymorphonuclear leucocytosis of from 15,000 to 30,000 per cubic millimetre, and moderate eosinophilia is not uncommon. The erythrocyte sedimentation rate is usually high. Albuminuria with persistent red cells in the urine is frequent, and casts of various types are occasionally present. The blood urea may be raised, and this may be very high as a terminal event. Blood cultures are always negative.

Local

Local manifestations are often widespread and bizarre, with fresh lesions occurring in one region and healing in another.

Abdominal.—Symptoms vary from minor colicky pains to what is apparently an acute abdominal emergency, when extensive mesenteric thromboses may have occurred. Epigastric discomfort is common, as is anorexia and vomiting. Haematemesis or melaena may be presenting features. There may be transient

abdominal tenderness and the liver and spleen are often enlarged Occult blood may be found in the stools, without clinical symptoms

Pulmonary.—Pleuritic pain, cough and haemoptysis are common, transient physical signs may be found in the chest Pleural effusion is rare Skiagrams may show transient opacities suggestive of infarctions

Renal.—Attacks of lumbar pain and haematuria are among the most frequent renal symptoms Transient albuminuria and the passage of red cells in the urine may be observed without obvious cause *Hypertension with uraemia is a* common termination of the disease, and a proportion of such cases are wrongly labelled ' nephritis "

Cardiac.—Tachycardia dyspnoea and praecordial pain are common symptoms, the heart may be enlarged and typical myocardial infarction may occur Transient pericardial frictions may be observed As renal involvement is so prevalent, *the presenting features may be hypertension and cardiac hypertrophy*

At post-mortem examination the heart is frequently involved even when symptoms have been minimal during life

There is nothing characteristic in the electrocardiographic tracings, although transient minor abnormalities may be seen and gross myocardial infarction is not rare

Neurological.—Recurrent severe headaches, painful stiffness of the neck and peripheral neuritis are perhaps the most common neurological symptoms The cerebral vessels are less often involved than elsewhere, but focal lesions occur in any part of the nervous system Hypertensive encephalopathy may be a feature in the later stages

Locomotor.—Transient arthritis, myalgias and tender nodules here and there about the body are frequent manifestations

DIAGNOSIS

The clinical picture of a chronic febrile wasting disease, with disseminated lesions about the body, is characteristic if polyarteritis nodosa is kept in mind as a possibility

The following conditions have to be considered in the differential diagnosis

Subacute bacterial endocarditis.—In this condition the Osler s nodes, petechiae and cardiac murmurs, together with a positive blood culture, are diagnostic Hypertension is rare in this disease

Tuberculosis chronic disseminated or renal.—In this the skiagram of the chest may show infiltration or enlarged mediastinal glands a polymorphonuclear leucocytosis is uncommon and *Mycobacterium tuberculosis* can generally be recovered by direct smear or culture from gastric secretions, urine or blood

Sarcoidosis.—This condition perhaps most closely simulates polyarteritis [illegible] there may be [illegible]

in the lung fields, with [illegible] lesion, or failing these, a biopsy of liver or tonsil may show the characteristic histology

FIG 36 — Temporal or giant cell arteritis Left temporal artery showing gross intimal thickening composed of cellular fibrous tissue with mucoid material between the cells Segments of the internal elastic lamina are destroyed with fragmentation of remaining portions The whole circumference of the media is involved in varying degree with areas of coagulative necrosis and heavy cellular infiltration Multinucleated giant cells of the foreign body and Langhans type are seen in relation to the fragmented internal elastic lamina The adventitia shows a cellular infiltration of lesser extent Haematoxylin and eosin × 39 (reduced by ⅓ on reproduction) (*By courtesy of Drs B S Cardell and T Hanley and the Editor of "J Path Bact"*)





FIG 37 — High power of Fig 36 showing outer zone of intima and media Cellular infiltration and giant cells Haematoxylin and eosin × 175 (reduced by ⅓ on reproduction) (*By courtesy of Drs B S Cardell and T Hanley and the Editor of "J Path Bact"*)

History Male aged 68 years Myocardial infarction when aged 55 years and 61 years Symptoms began four months before death with generalized pains in neck shoulders and calves and with anorexia and constipation After this he developed excruciating pain in the right neck and tempero-parietal region followed in a few days by pain in the left temporal region After eight weeks he complained of diplopia followed in a few days by blindness of the left eye with occlusion of the central retinal artery He became completely blind with similar changes in the right eye a few weeks later He later developed dysphagia and dysarthria and died about sixteen weeks after the onset of symptoms

Presenting clinical features were low grade pyrexia tender thickening of the temporal arteries and a high erythrocyte sedimentation rate

CHAPTER 8

ASTHMA

E R BOLAND

DEFINITION

ASTHMA is the name given to difficulty in breathing of a paroxysmal wheezing type, which is apparently due to diminution in the bronchial lumen and may be caused by bronchial obstruction, muscular spasm, vascular engorgement, oedema or increased secretion

It is, therefore, the name for a symptom produced by widely different conditions, and not strictly for a disease The term is used, for example, in connexion with cardiac disease, in which case it would appear to be caused by temporary engorgement of the lungs when the left ventricular output does not keep pace with the right ventricular input It is also used in connexion with the dyspnoea of advanced renal disease, in which the causes may be partly cardiac and partly metabolic, and it can be used in connexion with the postural dyspnoea produced by the pressure of mediastinal tumours In this section, however, it is proposed to deal only with what is often termed bronchial asthma, which is paroxysmal in nature and in which, between paroxysms, the patient is subjectively and objectively in good health

Compilers of text-books of general medicine are often at a loss as to the proper section for allocation to asthma It is variously allotted to the sections on diseases of allergy, diseases of unknown origin, diseases of metabolism, diseases of the respiratory tract and disorders of the psyche—all with more or less justice It can, in the absence of any general aetiological certainty, be justly dealt with in a text-book on respiratory diseases, since, in a condition where so much else is uncertain, it is at least incontrovertible that most of the symptoms and complications of asthma are respiratory Although more tons of paper and more gallons of ink have probably been expended on explaining the aetiology of asthma than have been expended on any other common condition, we still know little of the true causation of asthma This is a statement which would not be accepted by allergists, who have few doubts either about the cause or the treatment of bronchial asthma Thus Unger (1945), in his text book, *Bronchial Asthma*, states that, the words bronchial asthma are to be employed to mean Allergic Bronchial Asthma , he also states that therapy is now based on three fundamental principles (1) the discovery, if possible, of the causes of the attacks, (2) the elimination of the cause, or (3) the desensitization of the patient if elimination is impossible

This simple conception of asthma and its treatment is to be found in many text-books, and is the basis of most of the treatment by allergists , it is believed in by many practitioners and even by many patients, but as a conception it is over-simplified and the treatment is over-rated

ALLERGY IN ASTHMA

Allergy is a vast subject and there is no space here to deal with it in detail, but the term allergy meaning altered reactivity was coined by Von Pirquet in 1906 to describe the changes in tissue reaction occurring in some cases as the result of initial injections described first by von Behring Richet and others This term has been used to describe the sensitization reactions of man and it includes hypersensitivity idiosyncrasy sensitization and anaphylaxis

The allergic theory of asthma is that certain individuals are or become sensitized to substances which are not normally harmful to normal individuals These substances are called allergens and most of them contain protein which is regarded as most probably the active portion of them It has also been said that some polysaccharides and some lipoids may have the same effect Some drugs which produce allergic responses do not contain protein The exact mechanism of the production of allergic responses is not really known although the theory is that they act by producing antibodies in the allergic subject and that the combination of allergens with fixed antibodies in the tissues produces the allergic response possibly by the release of histamine or some allied substance The nature of the response depends on the site of the allergen antibody reaction in the skin urticaria or oedema in the nose rhinorrhoea in the intestine colic possibly due to muscular spasm or intestinal urticaria or both in the bronchi broncho-spasm and oedema and hypersecretion by the mucous membranes

The exact mechanism by which the reactions are brought about is uncertain but the probability is that in the case of bronchial asthma the afferent paths may be from the nose or from the bronchi themselves or possibly from the stomach or other organs and that the action is by vagal stimulation with consequent constriction of the smooth muscle of the bronchi and hypersecretion

There is plenty of evidence for considering a great number of cases of asthma as being in the allergic category For example in a considerable percentage of cases there is a family history not only of asthma but of many allied disorders considered to be allergic in origin such as hay fever urticaria food sensitivity and less certainly migraine Furthermore asthmatics are themselves notoriously sensitive to injections of serum and especial care has to be taken in giving them therapeutic injections of proteins or even in the administration of drugs When proteins are injected into the skin of asthmatic patients they cause local reactions to certain of the substances injected which are quite different from the reactions to others or to the reaction of controls The skin reaction and sometimes the attacks of asthma are associated with a local or general eosinophilia which is one of the concomitant factors in allergic manifestations as a whole

Clinically cases of asthma are seen in which the patient can be shown to be sensitive to simple substances such as grass pollens which can cause both hay fever and asthma in the same subject others only get their attacks when exposed to the inhalation of certain dusts at their work and never at other times Asthma in many cases can be proved to be provoked by exposure to horses dogs or cats to feathers to kapok to face powder to house dust and to a whole host of simple and complex substances To the allergist the whole thing is simple allergen + antigen = asthma Without allergens without antigens there can be no asthma

There is no question about the importance of allergy in asthma, but perhaps too much emphasis has been placed on it *through concentration on* the anti-allergic methods of treatment which have pre-occupied so many professionally successful allergists, so that what lies behind the allergy has often been ignored

OTHER AETIOLOGICAL FACTORS

Psychological factors

There is no doubt whatever of the psychological aspect of asthma, even if one cannot go as far as some psychiatrists, who regard asthma as a psychoneurosis and the allergic manifestations as secondary *to the psychoneurosis Some* psychiatrists have claimed to be able to cure their patients by psychoanalysis alone without bothering about the allergens at all Allergists agree that psychological treatment is important, but only as a secondary factor in a condition in which the allergens play the leading and essential role

Other mechanistic theories

There are other mechanistic theories, such as the one which suggests that there are trigger zones in the nasopharynx, which can produce attacks of asthma when stimulated, and that, as a natural consequence of this, the patients can be made insensitive by operation, by cauterization or by local injections, but these have fallen into disrepute Other authorities state there are biochemical or endogenous toxic causes for asthma , this theory leads to elaborate dietetic therapeutic programmes There is also an infective theory of the causation of asthma These and other theories are referred to below

Extrinsic and intrinsic causes

Rackeman (1931) neatly classifies the types of asthma into (1) extrinsic asthma, in which he includes *all the asthma produced by extrinsic allergens inhaled,* ingested or injected, and (2) intrinsic asthma, into which are bundled all the other sorts—infective, toxic, neurotic, reflex and all the rest Many cases, however, refuse to stay neatly even in these comprehensive pigeon-holes, and many cases that might reasonably be classified as extrinsic are affected or modified by *causes that should be labelled intrinsic However, as the classification* has some slight bearing on treatment, it may serve a useful purpose

Incidence

[illegible] frequency of the condi- [illegible] ice suggested that the [illegible] 1 in every 170 (0 6 per cent) Study of groups in Great Britain and in the United States of America and elsewhere suggests that an incidence of *about 0 59 per cent is probably correct* for both this country and the United States

Race—Little is known about racial susceptibility except that it is probable that the disease is much more common in the "civilized" races than in the others, and that Jews have a relatively higher incidence as a racial group

Sex—There is approximately the same distribution of asthma between the sexes, although some writers state that it is slightly more common in males

Age—Asthma makes itself apparent in the first decade of life in approximately 30 per cent of cases; most of the other cases are fairly evenly spread over the next three decades Excluding cardiac and renal asthma, the disorder is much more rare after the age of 50 years, but it does occur at this age and even later Those who classify asthma as extrinsic or intrinsic would say that extrinsic asthma is much commoner in younger people, while intrinsic asthma is much commoner in the older group

Social status—Asthma is rather more common in the relatively well to-do, and amongst the workers it is more common in sedentary than in manual workers, even making allowances for the special exposure of these latter in particularly dusty trades, such as flour-millers, cloth workers, rag sorters, furriers, hairdressers, poultry-dealers and the like

Intelligence—There is a fairly general idea that a high proportion of asthmatics are to be found amongst the more intelligent section of the community There may be some truth in this, but the sufferers from asthma are often more aggressive, more self assertive and emotionally less stable than their non asthmatic associates They sometimes become extremely possessive, selfish and introverted, but most of the characteristics described may occur as the result of their being set apart by their condition and be otherwise unassociated

Environment—Geography plays little part in the aetiology of asthma No locality is completely immune Patients often attach great importance to environment, stating that they always get asthma when they go to X and never when they go to Y Occasionally there may be some particular exposure to allergens at X, which is absent at Y, but more often the attacks at X and the freedom at Y are the results of auto-suggestion At all events, in a large clinic, some patients will tell you that if they could leave X and be at Y all would be well, but others tell you that if only they could leave Y and go to X they are sure they would be "all right" Taking it by and large, patients are often freer from attacks at high altitudes, provided that they have not developed complications This may be due to the absence of dust, to the freedom from infection or the general well being induced by altitude, or to just plain suggestion

Heredity—The first book on asthma published in England, that by Sir John Floyer written in 1698, mentions the hereditary tendency in asthma Since his time there have been many studies, most of which tend to show that the family histories of asthmatics show others suffering from asthma or allergic diseases in approximately 50 per cent of cases, whereas controls give a family history in only about 7 per cent It would appear that more members of the family are affected when both the parents have an allergic history than when only one has this, and it would appear in the majority of cases that what is transmissible is the asthmatic diathesis and not sensitivity to any particular substance, although there are family histories which appear to show a transmissible hereditary sensitivity to eggs, strawberries or other foods

In general, asthmatic subjects tend to occur in small families, and genetic studies do not reveal a convincing mendelian pattern

CLINICAL FEATURES

Description of asthmatic attack

Precipitating causes—Asthma may occur at any time of the day, but in mild cases it commonly starts in the early morning and wakes the patient up, or else it may become more obvious when the patient wakes at his normal time and is getting out of bed The stimulus which causes the attacks in the latter case is probably the first breath of cold air when the patient wakes up, associated with the movement and deeper breathing on waking which disturbs the secretion that has been collecting during the night Any patient with acute or chronic bronchitis starts to cough as soon as he gets up in the morning, and the colder the air he inhales the more prompt is the reaction In a case of ordinary bronchitis the result is coughing, in the asthmatic the result is bronchial spasm The probable explanation of the nocturnal attacks is more complicated Metabolic changes during sleep have been postulated but remain undiscovered Feather sensitivity, due to inhalation of dust from the pillows, has also been suggested, therefore many an asthmatic has been advised to sleep on kapok or, if he is sensitive to kapok, to sleep without a pillow or on a pillow covered with oiled silk or Cellophane, but the real explanation is probably a mechanical one—the result of respiratory embarrassment in the horizontal position, especially with the slight encroachment of the soft parts and interference with the air-way in deep sleep

Attacks are often produced by exertion, probably associated with increased ventilation due to exertion, and they may be precipitated by emotional factors They can, of course, be produced by exposure to specific inhaled allergens, such as, for example, certain pollens and industrial and domestic dusts Exposure to cold air is mentioned above, exposure to fogs, smokes or chemical fumes may have the same effect in asthmatics, and in such a case it is merely an exaggeration of the normal reaction in non-asthmatics

Nature of the attack

The typical attack varies from a slight feeling of constriction behind the sternum or trachea or around the ribs, produced only on attempting deep respiration or on exertion, to the most intense discomfort with violent, noisy inspiratory effort and expiratory distress During the severe attacks the patient is reluctant to move, he sits bolt upright in bed with his head thrust down and forwards, his mouth open and his alae nasi working, his chest is elevated, his shoulders are raised, with all his accessory muscles being used to move his chest, the veins in his neck are bulging, his sternomastoid muscles are taut, and his diaphragm is heaving Often he will lean forwards over a table or chair in order to breathe more freely

All this effort produces much respiratory movement, but it is rather an up-and-down movement than an expansive one If the attack is a prolonged one or the patient has become emphysematous as a result of repeated attacks, he will become cyanosed He can talk, if at all, only in gasps, is unable to eat or drink, and may, in some cases, become most upset and emotional His respiratory embarrassment is audible first as an expiratory wheeze and then by wheezing inspiratory and expiratory sounds, interrupted from time to time by a short, usually ineffective, cough

Objective findings

On examination it will be found that the expansion of the chest is grossly diminished even when the movement is maximal The rhonchi can be felt, the chest is everywhere hyper-resonant, the cardiac and liver dullness is decreased, and the diaphragm is depressed Auscultation shows that the breath sounds are diminished but are often obscured by sibilant and coarse rhonchi, in the later stages there are râles of all sorts of pitch, more or less uniformly distributed through the chest

That the physical signs of hyper-resonance are due to over-distension of the lungs with air, and the rhonchi mainly to bronchial spasm, can be shown by giving the patient an adequate dose of adrenaline After this medication, hyper-resonance disappears, the normal pulmonary boundaries are restored and the rhonchi disappear, in an early case In the more chronic cases the originally transient distension of the asthmatic attack results in permanent distension due to emphysema

Periodicity of attacks

Some cases show a periodicity which is of some interest Certain patients who are sensitive to pollen get asthma only during the periods when the pollen is air-borne and not at other times of the year A large group who suffer from bronchitis, either as the result of, or as the cause of, their asthma, only get attacks during the winter months and not during the summer Many patients with asthma gain complete relief or improve with puberty, but some cases appear for the first time at that period Some patients state that their attacks coincide with their menstrual periods, but some women tend to place all unusual phenomena in some relation to their periods, and there is little evidence that there is any real connexion, the menopause does not seem to affect the course of the condition Many more women are relieved of their attacks during pregnancy It is not clear whether the factors here are metabolic, mechanical or psychological Sometimes patients have attacks only at week-ends, or during school holidays or similar periods Here again the explanations may be multiple environment, change of habits or routine, topical exposure to allergens, or psychological causes may each or all be operative

Conditions allied to asthma

Hay fever.—Certain patients are sensitive to grass pollen or, more often, to grass and tree pollens These cause seasonal rhinorrhoea, sometimes associated with attacks of asthma The tendency is for the attacks to become less severe as the patient grows older, and they tend eventually to disappear spontaneously Records of this sensitivity frequently occur in the family history of cases of asthma

Allergic rhinitis.—The symptoms of this condition are similar to those of hay fever, namely sneezing, running from the nose, and nasal obstruction, but the disorder is not seasonal in duration and does not appear to be due to pollen It tends to occur in many people on first rising in the morning and, although it may remain bothersome for years, it tends to die out in the long run Many explanations have been given for it, varying from sensitivity to dust, or orris face-powder to local nasal conditions such as polypi or sinusitis It is certainly

commoner in women, particularly young ones Sufferers from this condition not infrequently tend to suffer from asthma sooner or later, and it also is an important finding in the history of asthmatics

Food idiosyncrasies—Some patients get acute abdominal pain after the ingestion of certain foods *All sorts of foods have been implicated*, but chocolate, strawberries and eggs appear rather more frequently in case reports of this condition than do other foods

Urticaria and angioneurotic oedema—These conditions are often allergic and are sometimes definitely associated with the ingestion of certain drugs or foods, or with the administration of serum but they may appear apparently spontaneously or, at all events without manifest cause These conditions tend to occur rather frequently in asthmatics but it does not seem that the reverse is as common

Migraine—This is often said to be allergic in origin and therefore might be of some importance in history taking but its exact relation to asthma is rather uncertain

Eczema and prurigo (Besnier s)—Eczema is often found in infants and young children, either associated with asthma or preceding it It is thought to be allergic in origin It rarely persists into adult life and it even more rarely appears in adult sufferers

Seasonal conjunctivitis—Conjunctivitis is often associated with hay fever but cases occasionally occur independently of the nasal symptoms

Other conditions—Many other conditions, from Meniere s disease to epilepsy, have been described as allergic and therefore important in an allergic history, but their position in this regard is, unlike the conditions mentioned above, more than dubious

Status asthmaticus—*See under* Treatment

Complications of asthma

Emphysema—Probably this is one of the commonest and most important complications If spasmodic distension of the lung occurs sufficiently often and for sufficiently prolonged periods, over distension and degeneration of the alveoli follow and the emphysema becomes permanent Most chronic asthmatics show evidence of this condition, and the well known barrel chest, with the hunched shoulders and the forward stoop is the typical appearance of the confirmed asthmatic When asthma occurs early in life, before the skeleton is fixed, there is often sucking in of the lower intercostal spaces and a pigeon chest develops

Loss of vital capacity—The vital capacity in asthmatics is considerably diminished, at first during the attacks with recovery afterwards, later the diminution becomes permanent, although it is surprising what an improvement in vital capacity may be induced by re education in breathing even in severe cases

Bronchitis—Bronchitis can be a complication of asthma, or else asthma can be a complication of bronchitis In most long standing asthmatic cases bron

[illegible] complicates these

to be secondary to bronchitis and often these cases start with what appears to be a common cold but the infection of the bronchi becomes chronic In many cases the upper respiratory catarrh is almost certainly a common cold although the possibility of allergic rhinitis must be borne in mind

Spontaneous pneumothorax —This condition is found occasionally when asthmatics are often and routinely submitted to radiography The probability is that the condition is due to the rupture of emphysematous bullae under the strain of the asthmatic attacks The symptoms are apt to be submerged in those of the asthmatic attack and the physical signs may be so submerged also so that the fact that the condition is found not infrequently in a casual manner suggests that it occurs more frequently than it is diagnosed Occasionally it is associated with surgical emphysema

Bronchiectasis —This condition is more often associated with asthma than appears in most text books and there is a generalized bronchial dilatation which gives a rather characteristic radiological picture with marked hilar enlargement and radiating opacities These cases are often erroneously diagnosed as pul monary tuberculosis because of a superficial resemblance in the radiological picture Not infrequently evanescent tension cavities are associated with this condition

Pulmonary tuberculosis —Asthmatics are not immune from pulmonary tuber culosis but when the disease occurs in an asthmatic the diagnosis is apt to be made rather late because all the symptoms and physical signs tend to be attributed to the long standing asthma emphysema and bronchitis

Cardiac complications —As a late complication chronic anoxic pulmonary heart disease (cor pulmonale) becomes an important and probably an invariable accompaniment of the chronic case in old age The condition is more likely to develop in cases complicated by the diffuse bronchiectasis described above

Massive collapse —Massive collapse of a lobe or lobes occurs occasionally in asthma when the partially constricted bronchioles are occluded by plugs of imper vious mucus This is most likely after the use of morphine and is the probable cause of death in some cases

PROGNOSIS

Considering the apparent severity of the symptoms and the discomfort caused by them it is surprising how little asthma seems to curtail the expectation of life of the sufferers A great many patients live on to a ripe old age even when the symptoms are continuous It is sometimes stated that no one ever dies of asthma but this would be an overstatement even if one excluded deaths from the admini stration of morphine or similar drugs and the occasional accidents of adrenaline administration since the emphysema which complicates the long standing condi tion may cause serious cardiac disability and there is always the possibility of broncho pneumonia as a complication Nevertheless even allowing for these possibilities it is surprising how rarely lethal is a condition at once so spectacular and so crippling

DIAGNOSIS

Clinical diagnosis

This is made almost entirely on the clinical history paroxysmal shortness of breath with tightness in the chest and wheezing, but with characteristic freedom from symptoms during the intervals between attacks The occurrence of the attacks in the early hours of the morning or on waking or at certain seasons of the year, and the relief of symptoms by antispasmodics such as adrenaline or ephedrine, the family history, and the personal history of associated conditions such as hay fever or eczema, all combine to present a picture which helps to differentiate asthma from other conditions somewhat similar in some respects The most difficult condition to differentiate, however, is cardiac asthma

Differential diagnosis

From cardiac asthma —The age of first onset is of some importance in differentiation Cardiac asthma would be unusual before the age of 45 years, except in the presence of valvular disease of the heart, which would be manifest It is unfortunately not safe to assume that nocturnal dyspnoea, coming on for the first time over the age of 45 years, is unlikely to be bronchial asthma, because, although asthma usually starts in childhood or adolescence, not a few cases, especially those associated with bronchitis, occur for the first time in the fifth decade or later The patient with cardiac asthma may have other signs of cardiac decompensation *but this is not essential He will usually suffer from more distress and anxiety* than does the patient with asthma from other causes His lungs will often show marked basal oedema and his sputum may be blood stained A case showing well-marked cardiac disease is easy to distinguish but there are borderline cases which are much more difficult

From constriction or obstruction —A rare cause of pulmonary wheezing is constriction of the bronchi, produced by enlarged glands or pressure of an intrathoracic goitre, or by an enlarged thymus gland or a bronchial or mediastinal neoplasm The wheezing is then often localized or unilateral and presents little difficulty in differential diagnosis

Diagnostic aids

Skin tests

These are performed either by scratching the skin through a drop of a solution containing an extract of various protein substances, or else by the intracutaneous injection of similar solutions By this means it is possible to test the sensitivity of the patient to a wide range of substances, first by testing for grouped reactions for a range of substances—for example, feathers or moulds—and then, if sensitivity is shown, by making differential separate tests on the various component parts of the group Some clinicians attach great value to these tests, and some text-books suggest that the whole diagnosis of the cause of a patient's asthma and his consequent treatment depend on the identification of the offending allergens by means of skin tests, and on the desensitization of the patient by the injection of small but increasing doses of the allergen in question It is a simple conception but, alas, far from the truth, for the skin tests do not tell a simple story

Reactions to skin tests are certainly more likely to be positive in patients who suffer from asthma or allied conditions, but they are often positive in a great many people who do not have these conditions or any family history of them. The reactions are not always quite consistent in the same patient. There is no certain correlation between the protein to which the patient's skin is sensitive and the cause of his asthmatic attack. Thus skin tests form an interesting study and are occasionally useful, but we are far from understanding their full significance and any considerable reliance on them for the guidance of treatment is unjustifiable.

Eosinophilia

Abnormal numbers of eosinophil cells may be found in the sputum or in the blood during attacks of asthma and may therefore be of some diagnostic significance; but in the majority of cases they are absent and their appearance seems to be more common in the first attacks than in later ones.

TREATMENT

Treatment can be divided into (1) preventive treatment, (2) symptomatic treatment and (3) specific treatment. Whole books have been devoted to the third division. For reasons which I mention below, not much time is devoted to it in this article.

Preventive treatment

General protective measures

Preventive treatment is any treatment which is designed to make the occurrence of attacks less likely. Patients, for example, may be advised not to take heavy meals last thing at night, since it has been found that a full stomach at bed time makes nocturnal attacks more likely; or they may be advised to avoid cold baths in the early morning or to avoid the stimulating effect of frosty air soon after they wake up.

Patients may be given sedatives regularly, such as phenobarbitone, either during the day time or, if their attacks are nocturnal, last thing at night, in order to make the attacks less likely. They may be given ephedrine regularly for the same reason whether they are having the attacks or not. They may be taught breathing exercises, such as those prescribed by the Asthma Research Council. These improve and maintain the mobility of the chest and the diaphragm, diminish the dead space and probably have a psychological effect on the patient as well.

Avoidance of allergens

Some people consider that the most important part of preventive treatment is the avoidance of allergens when possible. Patients who know that they are sensitive to dogs, cats, horses, hay pollen and the like should take steps to avoid them. Allergists interpret the skin tests as indicating the substances to be eliminated from the patient's diet or from his environment. They sometimes go to great lengths in this direction, making the patient wear special clothing and sleep in rooms denuded of all hangings or carpets, and telling him to use pillows covered by Cellophane or with their filling confined to flock or kapok instead of down feathers. Completely dust proof rooms have been constructed for these patients, but extravagant measures based on the uncertain premises of the skin reactions are

not justifiable It is always worth while to try the effects of eliminating substances to which the patient appears especially sensitive, and some success attends this attempt, especially in early cases in which the sensitizations are simple and few and multiple sensitization has not taken place As a general rule it is wise for a patient to avoid dusty atmospheres and to keep out of the way when dusty domestic operations are in progress, and the rooms in which he lives should be as clean and dust-free as good housekeeping should ensure

Occupation and environment

Advice which involves a patient in changing his occupation should not be given lightly, since an unsettling change may provide more symptoms than it is designed to relieve Sometimes it is better to keep a patient in an occupation which is theoretically unsuitable but which he enjoys and is good at, than encourage him to transfer to another in which he may be miserable and unsuccessful

On the same principle, changes of house or environment should not be light-heartedly recommended They are rarely successful, and in any large clinic, patients from town X will say that if only they could be in town Y all would be well, and at the same time patients from town Y say that if only they could live in town X all would be well With the rare exception of a simple sensitization to an allergic contact in one house and not in another, these odysseys are doomed to disappointment The indications for advising change of employment or environment are usually quite obvious and definite when the sensitization is single, proved and definite, which, in fact, is rarely the case

Symptomatic treatment

This is unfortunately, at the present time, in default of any real curative treatment, the most important section of treatment

Relief of bronchiolar spasm

The main object of therapy is the relief of spasm of the bronchioles

Adrenaline.—This is best achieved in the majority of cases by the use of adrenaline The drug can be administered in a spray, by the use of nor adrenaline tablets dissolved in the mouth or, most effectively, by the subcutaneous injection of 1 1,000 adrenaline Responsible adult patients should be trained at an early stage to use a hypodermic syringe and a solution of adrenaline The correct individual dose has to be arrived at by trial and error, but an in [illegible] s can be followed by the injection of appro[illegible], symptoms are relieved This [illegible] is effective in the majority of [illegible]ion of the dose obviates the side effects of [illegible], sometimes associated with large doses of adrenaline [illegible] The earlier the injection is given during the attack, the less is the quantity necessary and the more complete the relief Adrenaline should not be given intravenously since it may cause sudden collapse, deaths have followed accidental injection into a subcutaneous vein It is impossible to over-estimate the value of adrenaline in asthma and fortunately few adrenaline-resistant cases are seen Patients will sometimes say that adrenaline is of no help to them This is often found on re trial to be incorrect in fact, and to be due to incorrect administration in the past and the fixed erroneous conceptions

to which asthmatic patients seem to be peculiarly liable. Intramuscular injections of 1 : 1 000 adrenaline in oil can also be given. The object of giving it in this form is to produce a more lasting effect but it is rather disappointing in this context.

Cardophyllin (syn. aminophylline).—In the rare cases in which adrenaline does not produce relief, Cardophyllin (theophyllin ethylendiamine) given intravenously, is often effective in relieving bronchial spasm. It can be given in doses of 0·25 or 0·50 gramme of Cardophyllin in 20 millilitres of distilled water. Intramuscular injections are ineffective and painful. Cardophyllin can be given effectively *per rectum* in doses of 0·3–0·6 gramme in 15 millilitres of water. When it is given by this route there is a latent period of about an hour but the effect is more lasting than when it is given intravenously. In status asthmaticus Cardophyllin may be given continuously in saline solution in intravenous doses of 0·5–1·0 gramme in 1 000 millilitres of glucose saline solution.

Ephedrine.—Ephedrine sulphate is worth a trial in most cases of asthma. It can be taken either regularly or in advance of the time when attacks appear to occur. It can effectively be taken for example just before the patient goes to bed in order to prevent nocturnal attacks. It is wise in this case to combat the exciting and sleep-preventing effect of the ephedrine by giving ½ grain of phenobarbitone at the same time. Ephedrine although easy to take is unfortunately ineffective in many cases and a tolerance for the drug is often rapidly built up so that even if it is effective at first it becomes ineffective later. Intervals without ephedrine allow the recovery of its effectiveness in individual cases.

Histamine antagonists.—Phenergan, Benadryl and the like have proved disappointing in the treatment of asthmatic attacks and no antihistamine drug has yet been found fully effective in the control of asthma.

Inhalants.—Adrenaline can be given effectively by inhalation either from a simple vaporizing apparatus with a rubber bulb or else in more elaborate vaporizers using the passage of oxygen through the vaporizing fluid. The fluid usually used is adrenaline 1 : 100 solution. Isupren or Aleudrin, an iso-propyl derivative of synthetic adrenaline may be used instead. It has fewer side-effects than has adrenaline. There are many proprietary inhalant fluids on the market which are effective in so far as they contain adrenaline, the other constituents having little effect apart from this.

Hypnotics.—Hypnotics are often useful for asthmatic patients and the one most usually employed is phenobarbitone in doses of ½–1 grain. Morphine should never be given to asthmatics for the treatment of their asthma, particularly in status asthmaticus.

Re-training in breathing

Breathing exercises are a most effective form of treatment. They can be given to increase the vital capacity and diminish the dead space. In addition they may be successful as a form of psychotherapy. Breathing exercises often aggravate the shortness of breath and produce cough at first but they should be persisted in because ultimately they will give relief. It is advantageous to give inhalations of adrenaline before starting the exercises. The exercises should be taught in the first place by a physiotherapist skilled in their use. The exercises recommended are those published originally by the Asthma Research Council but which are

now published under the title *Advice to Asthmatics* by the Asthma Clinic (1949) at Guy's Hospital (London)

Treatment of status asthmaticus

Status asthmaticus—a condition in which the attacks become continuous, with little or no remission by day or by night—is the most alarming and most troublesome complication of asthma which the physician is called upon to treat. The consultant physician requires all the confidence born of his knowledge of the favourable prognosis in order to resist the impression produced by the suffering and anxiety of the patient and the terror and desperation of his relatives, as well as of the exhaustion and despondency of the patient's usual medical attendant. In the home, the long continuance of the attacks, in the face of every remedy, has often built up a psychological situation in which any rational approach to the handling of the case has become impossible. The patient is then best admitted to a hospital, preferably to a general ward. Matter-of fact reassurance of the patient, in a new atmosphere of apparent competence, induces an attitude of mind in him which is more favourable to suitable therapy. The presence of other patients besides himself who require attention removes him abruptly from the centre of the stage, which he has painfully occupied at home; the effect is often salutary and he begins to improve at once.

If the patient is cyanosed, he may with advantage be placed in an oxygen tent and given oxygen with 5 or 7 per cent carbon dioxide. The tent has the double advantage of supplying the needed oxygen and of screening the patient from others in the ward, who might be unduly sympathetic under the influence of the rather terrifying sight of his respiratory embarrassment, which, however genuine, is unconsciously exaggerated in the presence of an audience.

Adrenaline should be given by the minim-a-minute method and repeated at regular intervals if there is any response. These cases sometimes seem to be resistant to adrenaline; in that case, they are often relieved by intravenous injections of Cardophyllin, 0·25 gramme in 10 millilitres of distilled water; this treatment can be repeated 4 hourly until relief is obtained. Rectal administration of Cardophyllin (0·5 gramme in 20 millilitres of water) can be substituted for or combined with intravenous injections. The effect of intravenous injections should be immediate; the effect of Cardophyllin rectally takes about an hour to develop but the effect is more prolonged. If there is great dehydration, Cardophyllin may be given intravenously in a continuous drip, but patients sometimes associate an intravenous drip with imminent danger of death and the psychological effect may be bad. Patients should be encouraged to drink fluids in the ordinary way, and can usually be persuaded to do so if their fears about their imminent dissolution have been effectively combated. Mild sedatives may be given, such as phenobarbitone, Sodium Amytal or Nembutal in doses of ¼–1½ grains, but it is not advisable to "push" these or any other sedative drugs.

In cases which fail to respond to other methods of treatment, ACTH or cortisone may be tried. Good results are claimed by Zeller, Randolph and Rollins (1950), Howard and his colleagues (1950) and others. The recommended dosage of ACTH is from 50 to 100 milligrams per day divided into 6-hourly doses given for from 3 to 4 days. Some workers have found cortisone less effective than

ACTH but in this case the usual dosage is 200 milligrams per day divided into 6-hourly doses. These substances are given by injection but cortisone has also been given by mouth (Schwartz 1951). This method of treatment is well worth an extended trial. Herxheimer (1952) has reviewed the results claimed by various American authors in 133 cases treated and notes that 53 per cent became symptom free, 19 per cent were partly improved and 28 per cent were unimproved. No similar study of a relatively large group of patients has been reported in this country but the results in individual cases although occasionally encouraging have not been spectacular when objectively studied.

Morphine should never be given to these patients. It is very tempting to give it in order to produce mental relief and rest. Many doctors give it for this purpose and the patient survives in a great number of cases, but sooner or later a doctor who is in the habit of giving it will give it to a patient whom it will kill as certainly as if he were strangled which indeed he is by his own internal secretions. Anyone who has ever seen a patient gasping like a landed fish will realize on what a narrow thread life depends in such a case. Morphine depresses the respiration so that the patient suffocates.

Breathing exercises and postural drainage should be encouraged at an early stage in order to remove the secretions in the bronchial tree. Expectorants are sometimes given with advantage.

Bronchoscopic aspiration would probably be helpful but bronchoscopy of a possibly terrified patient is extremely difficult.

The prognosis as regards recovery from these attacks however distressing they are is good provided that the patient is not elderly and that pulmonary infection does not supervene. This possibility should be guarded against by the use of antibiotics.

Specific treatment

Most text books and all allergists state that asthma is a readily curable disease; thus Unger stated in 1944 "In comparison with other chronic diseases such as chronic arthritis and cardiovascular renal disease the results of treatment in asthma are usually brilliant. In those who do not obtain complete relief there is usually amelioration of symptoms, i.e. the attacks of asthma are milder and less frequent. Failures occur of course but in only a small minority and are often due to negligence on the part of the patient (or parent) or of the Physician." Urbach and Gottlieb in 1949 wrote "The large group of allergic asthma including that due to pollen is today readily amenable to treatment." Rackemann (as quoted by Cecil 1947) more conservative, more objective, states that "the hope of permanent relief depends entirely upon the discovery and the removal of the cause." With that statement I am generally in agreement since he was referring only to "extrinsic" asthma but how much more easily is this said than done!

Rackemann does not suggest that it is easy or that it can always be done but hundreds of enthusiastic and successful physicians say that it can. They suggest that the cause in the majority of cases can be discovered by a correct interpretation of the history and of the skin tests. They therefore require that every effort should

be made to avoid or eliminate the cause or allergen and, if that is impossible or unsuccessful, that the patient should be desensitized against the allergen That is, he should be given repeated small, but increasing, doses of the antigen until an excess of antibodies is produced in the blood, so that further doses of antigen are immediately neutralized before they can have an effect on the tissues Since this conception would mean the continuous administration of antigen to keep up the antibodies to the desired level, it is further postulated that continual saturation with antigens, or the production of mild anaphylactic shock, eventually leads to permanent "de allergenization" through the prevention of the formation of antibodies Whatever the theory may be, the commonest treatment in vogue in extrinsic asthma is desensitization, specific or non-specific

Specific desensitization

Specific desensitization is carried out by the administration of preparations of the allergen or allergens considered to be the cause of the asthma in the particular case Allergists depend on the history and skin tests for the indications of the appropriate solution of allergens to be used, and the preparation is individually made up from these, but solutions are often prepared from a solution extracted from ordinary house dust if other specific indications are lacking Some allergists take a tremendous amount of trouble in trying to find an individual solution which is more likely to be effective, others depend on more or less stock solutions with minor additions Whatever the method used, good results are claimed and, what is more, are claimed by patients

I am dealing in cavalier fashion with a method of treatment about which many books could be and have been written, but before discussing it further I shall quote one piece of work which shows how difficult it is to be certain of cause and effect in such an uncertain condition as asthma The results of treatment by desensitization may be judged in the light of it

A few years before World War II, Witts and Conybeare, at Guy's Hospital in London, started some control experiments on the treatment of asthma, and the work was carried on by myself and Lintott up to the outbreak of war (Boland and Lintott, 1939)

A careful selection of cases was made, and the eventual series of about 50 cases in each group were the product of a careful screening of hundreds of patients No patient was selected for the series whose attacks were infrequent or were of recent origin, who had a history of long periods of remission for any reason, or who was unreliable in any way A careful and detailed history was then taken, giving a close account of the attack pattern, its frequency, duration, severity and causation together with the history of treatment, symptomatic and otherwise Such patients as gave a good history and were considered consistent and reliable were then subjected to routine investigations

They were advised to eliminate or avoid any allergens which seemed (on the history or, less certainly, on the skin tests) to be possible causes of their attacks They were given advice about the uses of the various symptomatic drugs, as mentioned above They were given general advice about eating at bedtime, avoiding dusts and so forth, and then were sent for 2 months' (or more) instruction in

breathing exercises At the end of this 3 months period a further assessment was made and it was often found that general treatment and advice of this sort had apparently had a good effect Therefore many cases had to be removed at this stage from the test groups originally selected However the few survivors were a well tried band whose symptoms still remained and whose pattern was still consistent

These highly selected persons were then divided into two equal groups One group was sent to a well known allergist for treatment by protein desensitization and the other group was treated by us with injections of normal saline solution After 3 6 and 9 months the cases were re assessed Of the number of patients sent for desensitization nearly two thirds had improved either objectively or subjectively These results considering the severity of the cases chosen for a study were striking but the results in the equivalent group treated with normal saline were equally good or even in our series slightly better

A further study of the groups showed what to me was the most important finding of the experiment I mentioned above that many patients were objectively or subjectively improved Those regarded as objectively improved were the ones whose improvement could be recognized by us from a study of our case records Those said to be subjectively improved—and these were the majority—were the patients who claimed to be improved although the improvement could not be substantiated from the case records For example a patient might claim to be much better since starting the treatment stating that he now only had attacks once a week Reference to the notes taken before the treatment commenced might give the frequency of attacks as being once a week When this was pointed out to the patient he would express surprise and say that anyway they only lasted about a couple of hours Reference again to the notes would show that according to the notes the average duration of an attack was about 2 hours The patient would be momentarily nonplussed when confronted with this history and would then say that at all events he was miles better than he had been Beyond this it was impossible to go

Now it is not perhaps really the business of a physician to argue with a patient who thinks he is better and if asthma is regarded as merely a symptom and the patient thinks he is better it could be maintained that he is better The fact is however that in these experiments normal saline solution proved as effective as were protein solutions intended to desensitize This gives food for thought but it may be reasonably stated that it is no valid argument against desensitization since the selection of cases and the solutions used may not have been the correct ones Even if we agree with this point of view though with some mental reservations the most important result of the investigations was the demonstration that the subjective improvement experienced by the patients was not susceptible of objective proof If effective desensitization were in fact frequently achieved by these methods of treatment improvement would not be a matter of opinion and a subject for dispute

Similar experiments were carried out in the controlled treatment of rhinorrhoea and the same results were achieved Just as many cases were relieved by normal saline solution as by protein solutions Here the question of subjective

improvement did not arise for the only criterion was the stoppage or otherwise of rhinorrhoea

Similar experiments were carried out with autogenous and stock vaccines, again with similar results The outbreak of the war prevented the extension of these experiments to hay-fever desensitization Rendered somewhat critical by our findings, we were anxious to see whether this treatment when critically reviewed, was any more effective

I have discussed these experiments at some length in order to explain why I have not devoted more space to what is a generally accepted treatment for extrinsic asthma, and I shall conclude by giving my views on treatment by desensitization with protein solutions and, incidentally, on the treatment of asthma with vaccines, autogenous or otherwise

Most treatment of this type is effective only by suggestion and the improvement produced is often more apparent than real If the effectiveness of the treatment is judged by "cures"—which may be a harsh criterion, but not an unreasonable one—the claims made by allergists are for 20 per cent "cure" The fact that the same figure can be produced by injections of normal saline solution suggests that the "cures" are the result of chance, of the uncertain natural course of asthma and of the powers of suggestion The theory of desensitization is a sound one but in the vast majority of cases not a practical one, possibly because the basis of asthma is the tendency to become sensitized, and because the majority of patients have become or are capable of being sensitized to so many different unavoidable substances that desensitization is difficult or impossible I still think that, if a patient can be found to be sensitive to one or two proteins only, it is possible that desensitization may be effective, but this slight residual speculative belief does not seem a firm substance for the treatment of allergic conditions, with which Urbach and Gottlieb, for example, deal in a book of nearly 1,000 pages

My attitude and theirs may perhaps be considered in the light of a paragraph in their book

"In conclusion, one more important point must be considered The great majority of physicians are of the opinion that asthma is a dangerous but practically an incurable disease Although they naturally refrain from expressing this opinion to the patient, the latter very soon senses this pessimistic attitude which very often does immeasurable harm psychologically and consequently physically The physician who is convinced that many cases can be cured and that almost all can be considerably improved and who is able to communicate this conviction to his patient, possesses the most important pre-requisite for success"

CONCLUSIONS

My scepticism with regard to the effects of present methods of so-called "specific" therapy may have conveyed the sense of pessimism which Gottlieb and Urbach rightly deplored I am not in the least pessimistic about the treatment of asthma from a symptomatic point of view A great deal can be done for most patients suffering from asthma Alteration of the patient's psychological reaction to his condition—and, as necessary, the alteration of the relatives' reaction to it—is of the utmost importance This can be done by explanation and reassurance

in most cases The use of breathing exercises and the intelligent use of antispasmodics have, in most cases, an excellent effect, and the patient gains in confidence when he knows that he has the means of relief at his disposal If it is possible to detect the precipitating cause of the attacks of asthma, and if this can be avoided, then the prognosis is so much the better I would not even exclude the possibility of desensitization in an individual case, if a single protein allergen appeared to be specially implicated I think that its effect is not proved, but that it might be worth a trial, although, in the vast majority of cases, I believe that present methods of desensitization have only a psychological value

If the attacks of asthma cannot altogether be eliminated, the patient can be taught to live with his asthma, just as a patient can be taught to live who has lost his arm or his leg He should at all ages be encouraged to lead a life which is normal so far as humanly possible , he should not be encouraged to regard himself as an invalid and, especially, not to regard his asthma as a justification for dodging responsibility or for getting his own way The power of the spirit is never so beautifully displayed as in asthma One man with quite mild asthma will mentally surrender and become a complete invalid , another with infinitely more severe asthma will struggle on and lead a useful and effective life in spite of his severe handicap Sir Arthur Hurst, a life long sufferer from asthma, who had undergone almost every treatment known to man without much effect, almost never allowed his symptoms to interfere with his physical or intellectual life He would do his ordinary ward rounds on a day and in a condition when other men would have been tucked up in bed, surrounded by anxious relatives and doctors

It is impossible to over-emphasize the psychological approach to the control and treatment of asthma The intelligent patient is often well aware that his symptoms are aggravated or produced by difficult psychological conditions To be aware of this is half-way to being protected against their effects The unintelligent patient is not aware of it, and may be incapable of being taught a proper psychological approach towards his condition It is in these cases that the impressive " mumbo jumbo " of some specific treatments is most effective, for a time

In the course of this article I have referred to various theories of the causation of asthma and have rather tended to accept the differentiation into extrinsic (or allergic) asthma and intrinsic asthma It would, however, be better to use the word, allergic, in a general sense, meaning an altered reactivity to various stimuli, which may be allergic in the sense of the allergists or may be psychological, reflex, metabolic or mechanical In that sense all asthma is allergic, and it conforms with Hurst s definition of the asthma diatheses (Hurst, 1943), which involves the inherited or acquired possession of an irritable bronchial system, including the medullary centre, the vagal nerve endings and the bronchial musculature and mucosa, which can be stimulated by blood-borne irritants and by reflex and psychic stimuli

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CHAPTER 9

EMPHYSEMA OF THE LUNGS

RONALD V. CHRISTIE

PULMONARY emphysema has been defined as a condition in which the alveoli of the lungs are dilated and their walls over distended. When used in this wide sense the term includes four clinical entities which are probably quite distinct: (1) chronic obstructive or hypertrophic emphysema, (2) senile or atrophic emphysema, (3) acute vesicular emphysema, and (4) localized or compensatory emphysema. There is also the rare condition of acute interstitial emphysema.

CHRONIC HYPERTROPHIC OR OBSTRUCTIVE EMPHYSEMA

Synonym: chronic vesicular emphysema.

This is the most common form of emphysema and is the type usually referred to when a diagnosis of emphysema of the lungs is made.

Morbid anatomy

Gross changes

The thoracic cage is usually enlarged with a characteristic increase in the antero-posterior diameter, giving the so-called barrel shaped chest. There is often a moderate degree of kyphosis involving all the thoracic vertebrae. The vertebral cartilage may be thin and compressed anteriorly where there may be lipping. Partly as the result of kyphosis the ribs run horizontally and are widely spaced and their cartilages frequently show ossification or become elongated and less elastic.

The lungs are voluminous and fail to retract when the thorax is opened. The surface of the lung may present a corrugated effect, the result of rib indentation, and the visceral pleura is thin, flimsy and atrophic. Bullae due to the protrusion of greatly distended alveoli are frequently seen. The lungs are dry and light, but they pit on pressure, not because they are oedematous but because they have lost their elasticity.

On section many of the alveoli can be seen to be dilated and their walls are thin and in places ruptured so that differentiation of the lobules into alveoli, atria and alveolar ducts is impossible. Because the alveolar walls become thin and disappear the air sacs may lose their honeycomb structure and so be deprived of their supporting framework; this is especially true of the surface of the lung, where large bullae may be formed. It has been claimed that the rupture of the alveolar wall begins at the so-called alveolar pores, but whether or not this is true seems to be of little significance. Of much greater importance is the effect of this destructive process on the air sacs of the lung and several changes have been described. The normal lung lobule, or air sac, consists of an alveolar duct opening into the atrium around which are placed the alveoli, and in health there is a sudden transition from

narrow alveolar duct to wide atrium, so that the inspired air may be thrown as a jet into the more remote parts of this terminal segment These lobules have been studied in detail by means of serial sections and of specimens injected with celloidin, and in emphysema it has been shown that the lobule is distorted and sometimes distended (Beitzke, 1925, Kountz and Alexander, 1934) The alveolar duct is dilated and funnel-shaped, so that air is no longer thrown into the atrium as a jet If it could be shown that this jet of air does, in fact, serve a useful purpose in ventilating the lobule, it would be reasonable to suppose that this function would be impaired in emphysema

Changes in the lumina of the bronchi and bronchioles also occur, but these are not constant The terminal bronchioles, as well as the alveolar ducts, are usually dilated, and this dilatation may extend up to the smaller bronchi, producing a picture resembling cylindrical bronchiectasis The state of the medium and larger bronchi is, however, very variable, and depends rather on the degree of bronchitis than on the degree of emphysema, although some atrophy and dilatation are usual in severe cases

Blood vessels

The capillaries may appear to be narrow or obliterated and sclerosis of the pulmonary arterioles is common There is often hypertrophy of the right ventricle, also enlargement of the pulmonary artery and, in advanced cases, right heart failure Not infrequently this is associated with hypertrophy of the left ventricle, which can usually, but not always, be ascribed to hypertension

Distribution of changes

The distribution of emphysematous changes is characteristic They are most marked in the superficial parts of the lung, particularly at the apices and along the margins In the early stages they may be confined to these areas but, as the disease progresses, emphysematous changes may be found throughout the lung

Loss of elasticity

Changes in the elasticity of the emphysematous lung were first suggested by William Watson in 1795 When the thoracic cage is opened on the post mortem table, the lungs do not collapse, and this alone would appear to be proof that the normal elastic recoil of the lung had been lost However, more detailed observations on the pulmonary elasticity, made by measuring the force required to inflate and deflate the lung have given conflicting results in emphysema (Tendeloo, 1925, Loeschke, 1928, Thies, 1932) Considerable changes in the elastic properties of the lung occur after death, and the significance of post mortem measurements of elasticity has been seriously questioned The fact that the lungs do not collapse when the thorax is opened has been said to mean only that they are over stretched in the same way as a spring may be overstretched and yet maintain much of its elasticity Histological study of the lungs gets us no further, as there is the same disagreement among morbid anatomists as to whether the elastic fibres are destroyed or merely overstretched, nor do the morbid appearances indicate whether loss of elasticity is an early or a late manifestation of the disease That loss of elasticity may be complete in this disease has, however, been shown by measurements of the intrapleural pressure during life (*see* Figs 38 39) The intrapleural pressure is an

indirect measure of the tension or elastic recoil of the lung and in emphysema it is not always negative as it is in health even at the end of a full inspiration the intrapleural pressure may be the same as that of the atmosphere and it is not altered when the lung is collapsed by a pneumothorax (see Fig 39) This can only mean that in emphysema the elasticity of the lung may be impaired at a comparatively early stage of the disease (Christie, 1934)

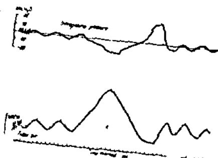


FIG 38 — The elasticity of the normal lung Synchronous tracing of tidal air (lower curve) and intrapleural pressure (upper curve) in a patient with a minimal tuberculous lesion at the right apex To make the tracing comparable with that shown in Fig 39 the lung has been partially collapsed by a pneumothorax so that the pleural pressure fluctuates around that of the atmosphere At A a deep breath is taken maximal distension of the lung corresponds closely to the time when the intrapleural pressure is most negative The elasticity of the lung is intact (*From Christie and McIntosh 1934*)

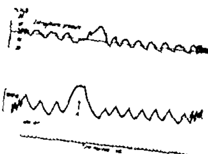


FIG 39 —Reduced elasticity of the emphysematous lung Synchronous tracing of tidal air (lower curve) and intrapleural pressure (upper curve) in a patient with emphysema and a pneumothorax of 40 cubic centimetres on the right side The pleural pressure fluctuates around that of the atmosphere At A when a deep breath is taken the pleural pressure becomes only slightly more negative and at the end of inspiration it returns to that of the atmosphere The lung does not begin to deflate until the pressure becomes positive These changes indicate that the elasticity of the lung has been lost (*From Christie 1934*)

These observations on lung elasticity are not merely of academic interest, for many of the anatomical and functional changes found in emphysema can be explained on the basis of its loss It is usually assumed that any loss of elasticity is secondary to over-distension What is not generally realized is that the converse is more likely to be true the enlargement of the air sacs and the fact that this enlargement is most conspicuous at the periphery of the lung can be explained as being the direct result of loss of elasticity That this is so is apparent if it is remembered that in health distension of the lung is maintained by traction of the chest wall and the diaphragm and that this traction is counterbalanced by the elastic pull of the lungs if the elastic recoil of the lungs is reduced or abolished as happens in emphysema the thoracic cage will tend to assume the inspiratory position and the lung will be distended It is probably this mechanism which is at least partly responsible for the enlargement of the chest and low position of the diaphragm in emphysema

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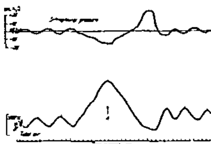


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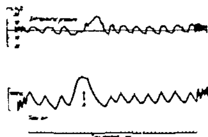


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chest wall and the diaphragm, and that this traction is counterbalanced by the elastic pull of the lungs if the elastic recoil of the lungs is reduced or abolished, as happens in emphysema the thoracic cage will tend to assume the inspiratory position and the lung will be distended It is probably this mechanism which is at least partly responsible for the enlargement of the chest and low position of the diaphragm in emphysema

narrow alveolar duct to wide atrium, so that the inspired air may be thrown as a jet into the more remote parts of this terminal segment These lobules have been studied in detail by means of serial sections and of specimens injected with celloidin, and in emphysema it has been shown that the lobule is distorted and sometimes distended (Beitzke, 1925, Kountz and Alexander, 1934) The alveolar duct is dilated and funnel shaped, so that air is no longer thrown into the atrium as a jet If it could be shown that this jet of air does, in fact, serve a useful purpose in ventilating the lobule, it would be reasonable to suppose that this function would be impaired in emphysema

Changes in the lumina of the bronchi and bronchioles also occur, but these are not constant The terminal bronchioles, as well as the alveolar ducts, are usually dilated, and this dilatation may extend up to the smaller bronchi, producing a picture resembling cylindrical bronchiectasis The state of the medium and larger bronchi is, however, very variable, and depends rather on the degree of bronchitis than on the degree of emphysema, although some atrophy and dilatation are usual in severe cases

Blood vessels

The capillaries may appear to be narrow or obliterated and sclerosis of the pulmonary arterioles is common There is often hypertrophy of the right ventricle, also enlargement of the pulmonary artery and, in advanced cases, right heart failure Not infrequently this is associated with hypertrophy of the left ventricle, which can usually, but not always, be ascribed to hypertension

Distribution of changes

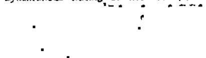
The distribution of emphysematous changes is characteristic They are mos[t] marked in the superficial parts of the lung, particularly at the apices and along th[e] margins In the early stages they may be confined to these areas but, as th[e] disease progresses, emphysematous changes may be found throughout the lung

Loss of elasticity

Changes in the elasticity of the emphysematous lung were first suggested by William Watson in 1795 When the thoracic cage is opened on the post mortem table, the lungs do not collapse, and this alone would appear to be proof that the normal elastic recoil of the lung had been lost However, more detailed observations on the pulmonary elasticity, made by measuring the force required to inflate and deflate the lung have given conflicting results in emphysema (Tendeloo, 1925, Loeschke, 1928, Thies, 1932) Considerable changes in the elastic properties of the lung occur after death, and the significance of post mortem measurements of elasticity has been seriously questioned The fact that the lungs do not collapse when the thorax is opened has been said to mean only that they are over stretched in the same way as a spring may be overstretched and yet maintain much of its elasticity Histological study of the lungs gets us no further, as there is the same disagreement among morbid anatomists as to whether the elastic fibres are destroyed or merely overstretched, nor do the morbid appearances indicate whether loss of elasticity is an early or a late manifestation of the disease That loss of elasticity may be complete in this disease has, however, been shown by measurements of the intrapleural pressure during life (see Figs 38–39) The intrapleural pressure is an

indirect measure of the tension or elastic recoil of the lung and in emphysema it is not always negative as it is in health , even at the end of a full inspiration the intrapleural pressure may be the same as that of the atmosphere and it is not altered when the lung is collapsed by a pneumothorax (*see* Fig 39) This can only mean that in emphysema the elasticity of the lung may be impaired at a comparatively early stage of the disease (Christie, 1934)

FIG 38 —The elasticity of the normal lung Synchronous tracing of tidal air (lower



At A a deep breath is taken maximal distension of the lung corresponds closely to the time when the intrapleural pressure is most negative The elasticity of the lung is intact (*From Christie and McIntosh* 1934)



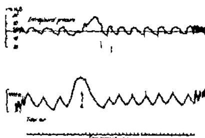


FIG 39 —Reduced elasticity of the emphysematous lung *Synchronous tracing of tidal* air (lower curve) and intrapleural pressure (upper curve) in a patient with emphysema and a pneumothorax of 40 cub c centimetres on the right side The pleural pressure fluctuates around that of the atmosphere At A when a deep breath is taken the pleural pressure becomes only slightly more negative and at the end of inspiration it returns to that of the atmosphere The lung does not begin to deflate until the pressure becomes positive These changes *indicate that the elasticity of the lung has* been lost (*From Christie* 1934)

These observations on lung elasticity are not merely of academic interest, for many of the anatomical and functional changes found in emphysema can be explained on the basis of its loss It is usually assumed that any loss of elasticity is secondary to over distension What is not generally realized is that the converse is more likely to be true the enlargement of the air sacs, and the fact that this enlargement is most conspicuous at the periphery of the lung, can be explained as being the direct result of loss of elasticity That this is so is apparent if it is *remembered that in health distension of the lung is maintained by traction of the* chest wall and the diaphragm and that this traction is counterbalanced by the elastic pull of the lungs if the elastic recoil of the lungs is reduced or abolished as happens in emphysema the thoracic cage will tend to assume the inspiratory position and the lung will be distended It is probably this mechanism which is at least partly responsible for the enlargement of the chest and low position of the diaphragm in emphysema

Loss of elasticity could also explain the over-distension of air sacs, and the bulla formation which occurs on the surface of the lung With loss of elasticity the expanding force is no longer equally distributed, and equal expansion in different parts of the lung should not be expected In fact, the greatest expansion should occur where the force is applied, and this is at the surface of the lung With each inspiration one would expect the superficial air sacs to be strained and stretched to a greater extent than are the air sacs deep in the lung, and, with this process going on for months and years, it is easy to understand the over-distension of air sacs and the bulla formation which occur mainly on the surface of the lung

Functional pathology

Haemo-respiratory exchange

The most important disturbance of function in emphysema is an inability to ventilate the blood, and this is usually apparent many years before the patient reaches the pathologist It is retention in the blood of carbon dioxide which is believed to be responsible for the dyspnoea on exertion that is an early manifestation of this disease cyanosis may appear later, and is due to deficient oxygenation of the blood Many explanations of this impairment of aeration of the blood in emphysema have been given, but most of them are based only on conjecture and can be shown to be unsound

Theory of muscular inadequacy —It has been suggested that the respiratory musculature is incapable of properly ventilating the lungs, for the same reasons that the vital capacity and chest expansion are diminished , but the burden of blame cannot be placed on the muscles of respiration, since ventilation of the blood is impaired even when the amount of air drawn into the lungs with each breath and during each minute is greater than normal Inability to hyperventilate on exercise may be a factor in the production of dyspnoea, but the main fault must lie in the lungs, because for some reason the air which is inspired does not properly ventilate the blood

Suggested barrier to the transfer of gases —It is obvious that the defect cannot be due to any barrier to the transfer of gases across the pulmonary epithelium, or to insufficiency of functioning lung, since, in these circumstances, the tension of carbon dioxide would increase in the blood but not in the alveoli The fact that changes in the blood are associated with proportionate changes in the alveolar air

1947) Although inability to increase adequately the cardiac output on exercise may be a factor in decreasing the exercise tolerance, it cannot be responsible for the impairment of gaseous exchange in the lungs

Inadequate ventilation of air sacs —A much more reasonable explanation, and one which is widely accepted on the Continent, is that the air sacs are not properly ventilated —partly because they are big, but mainly because the alveolar ducts are [illegible] Kountz and Alexander, 1934) It is true that in

has been named the "stratification" theory, so called because it assumes a gradient of ventilation in the lobule, those parts next to the alveolar duct being over-ventilated and the parts further away being under-ventilated. This sounds reasonable, but the validity of this theory of stratification depends on the assumption that in health there must be circulation of air in the air sacs and alveoli, and that diffusion alone is insufficient to maintain adequate exchange of gases in the lobules. The diffusion of gases is governed by well defined laws, and it can be calculated that, even if the lobule is 1 centimetre in diameter, diffusion alone would ensure the transport of gases across the alveolar space with a gradient of less than 1 millimetre of mercury. Unequal ventilation within the lobule cannot account for the extreme impairment of gaseous exchange observed in emphysema, in which the pressure of carbon dioxide may be raised by 30 or 40 millimetres of mercury (Christie, 1939; Rauwerda, 1946).

Increased volume of the lungs.—Increase in the volume of the lungs has been suggested as the cause of impaired ventilation. Provided that ventilation is distributed evenly throughout the lungs, an increase in lung volume does not significantly impair haemo-respiratory exchange (Bates and Christie, 1950).

Unequal distribution of ventilation.—An increase in the respiratory dead space has been suggested as the cause of impaired haemo-respiratory exchange, but there is ample evidence that any increase in the anatomical dead space—the space occupied by the respiratory passages down to the bronchioles—is too small to be of practical significance (Hoover and Gammon, 1915). I shall not enter into a discussion on the meaning of the physiological dead space. Most physiologists will agree that those who claim an increase in physiological dead space in emphysema are merely stating in a complicated way that the distribution of ventilation is unequal. In the normal individual the distribution of ventilation is remarkably even throughout the lungs, and consequently samples of alveolar air taken after expirations of varying depths are more or less uniform in composition. This is not the case in severe emphysema; the deeper the patient expires the higher is the carbon dioxide content and the lower is the oxygen content of the "alveolar air". These cases show the apparent paradox that, although the amount of air inspired is normal or even increased, many of the alveoli are under-ventilated (Roelsen, 1938 and 1939). Furthermore, the respiratory quotient of samples of air taken at the end of a forced expiration may be very low, and this can only mean that the sample has come from a part of the lung which is grossly under-ventilated. There is also ample evidence that the ability to produce a homogeneous mixture of gases in the lung is impaired in emphysema (Cournand and his colleagues, 1941; Bates and Christie, 1950; Briscoe, Becklake and Rose, 1951); this is due to over-ventilation of certain parts of the lung at the expense of other parts.

Pathological respiratory dead space.—The most likely explanation of this inequality in the distribution of ventilation is that much of the inspired air is deflected to the enlarged air sacs and bullae, the walls of which are avascular and which are therefore not in contact with the pulmonary circulation (Christie, 1939 and 1944; Riley and Cournand, 1949). This wastage of ventilation on functionless air sacs cannot be defined as wastage due to increase in physiological dead space, for there is nothing physiological about an emphysematous bulla. Nor can

it reasonably be called increase in anatomical dead space If a definition is wanted, I should prefer to give as the cause of impaired haemo respiratory exchange in emphysema the formation of a pathological respiratory dead space, consisting of bullae and other air sacs which are not in intimate contact with the pulmonary circulation In severe emphysema much of the inspired air is wasted by entering this pathological dead space, so that those areas of the lung which contain alveoli well perfused with blood are under ventilated The natural result of this abnormal distribution of ventilation in the lungs is imperfect aeration of the blood

Dyspnoea

The cause of dyspnoea in emphysema is clearly stimulation of the respiratory centre by the carbon dioxide retained in the blood The chronicity of the disease allows the patient with emphysema full scope for chemical compensatory changes in the blood, and the inability to eliminate carbon dioxide may be efficiently compensated by an increase in the bicarbonate reserve (carbon dioxide combining power) This compensatory mechanism is sufficient to ensure a normal acidity of the blood while the patient is resting, but any increase in metabolism—such as occurs during exercise or during a violent attack of coughing—is immediately associated with an increase in the blood carbon dioxide and stimulation of the respiratory centre

The emphysematous patient is, indeed in an unfortunate position Both inspiration and expiration have to be executed by unnatural respiratory efforts, and a considerable proportion of the air which is inspired is wasted by not coming into proper contact with the pulmonary blood At rest the patient can just make ends meet with the help of compensatory changes in the blood, carbon dioxide elimination and oxygen absorption keep up with the metabolic demands When the metabolic demands are increased by exercise, this balance breaks down The patient is unable adequately to increase the ventilation of the blood Carbon dioxide accumulates and anoxaemia increases, so that true chemical dyspnoea due to acid stimulation of the respiratory centre, appears In the absence of broncho spasm or heart failure, there is no orthopnoea because there is no pulmonary congestion which is the cause of true orthopnoea

The pulmonary circulation

Extensive changes in the pulmonary vascular bed can be seen at autopsy, and it is usually assumed that there is sufficient vascular destruction to impair the circulation through the lungs This belief is not based on any quantitative measurement, because it is not possible to estimate with the microscope even the approximate proportion of capillaries that have been destroyed It should not be forgotten that the pulmonary vascular bed has an enormous reserve, which is well illustrated in modern thoracic surgery, when the whole of one lung may be removed and yet there may be no evidence of impaired pulmonary blood flow or of strain on the right side of the heart A demonstration of capillary damage does not in itself, therefore, prove any embarrassment to the pulmonary circulation Much more suggestive of this are the changes frequently found in the pulmonary artery and right ventricle, as revealed by cardiac catheterization The pressure within the pulmonary artery is usually increased and this suggests increased resistance to the flow of blood through the lungs The cardiac output, however,

is also increased even under resting conditions, and the most likely explanation is that, under the stimulus of anoxia, the right ventricle maintains an increased cardiac output in spite of increased resistance in the pulmonary vascular bed (McMichael and Sharpey Schafer, 1944 , Richards, 1947) On exercise there is a further large increase in pulmonary arterial pressure but, in spite of this, the increase in cardiac output may be negligible (Hickman and Cargill, 1947 , Riley and his co workers, 1948) Inability to increase the cardiac output on exercise may be of considerable functional significance in emphysema , but these observations, which are recent, will have to be confirmed and amplified before they can be applied to the interpretation of symptoms in this disease

Electrocardiographic changes—Much has been written on the electrocardiographic changes in emphysema and in chronic cor pulmonale , there are right axis deviation and usually a prominent P-wave in lead II Praecordial leads may also show the changes characteristic of right ventricular hypertrophy (Myers, Klein and Stafer, 1948)

Lymphatic circulation—The lymphatic circulation in emphysema has received but scant attention The peripheral lymphatic vessels may show cellular infiltration, and the characteristic pigmentation of the lung may reflect deficient lymphatic drainage

The blood

The carbon dioxide combining power is usually increased in patients who are incapacitated by dyspnoea, and may be raised to over 100 volumes per cent This increase is a response to the retention of carbon dioxide, and represents the mechanism by which the acid–base balance is maintained , although the alkali reserve is increased, these patients are in a state of compensated gaseous acidosis The oxygen tension and oxygen saturation of the arterial blood are reduced Less commonly, in patients with severe cyanosis, there is polycythaemia

Riley and Cournand (1949) and other authors have recently emphasized the value of arterial-blood gas analysis in estimating the impairment of respiratory function in this disease Both the laboratory procedure and the interpretation of results in terms of "ventilation perfusion relationship" are complex, to say the least [illegible]

blood is in equilibrium with the carbon dioxide of the alveolar air, then the pressure of oxygen which should exist in the alveolar air and in the arterial blood, if a similar equilibrium existed with regard to oxygen, can be calculated from the respiratory quotient The difference between this "ideal" oxygen pressure and the oxygen pressure which is actually found in the arterial blood is expressed as the 'alveolar arterial gradient" This gradient is increased in emphysema and is assumed to reflect the passage of unaerated blood through the lungs It can also be expressed in terms of "venous admixture'

Clinical picture

History and progress

With rare exceptions there is a history of chronic bronchitis or asthma of several

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Riley and Cournand (1949) and other authors have recently emphasized the value of arterial blood gas analysis in estimating the impairment of respiratory function in this disease Both the laboratory procedure and the interpretation of results in terms of ventilation perfusion relationship are complex to say the least but the basis of this new line of approach is as follows The measurements which have to be made are the pressure of carbon dioxide and of oxygen in the arterial blood and also the respiratory quotient of the expired air If the assumption be made that in health as well as in disease the carbon dioxide of the arterial blood is in equilibrium with the carbon dioxide of the alveolar air then the pressure of oxygen which should exist in the alveolar air and in the arterial blood if a similar equilibrium existed with regard to oxygen can be calculated from the respiratory quotient The difference between this ideal oxygen pressure and the oxygen pressure which is actually found in the arterial blood is expressed as the alveolar arterial gradient This gradient is increased in emphysema and is assumed to reflect the passage of unaerated blood through the lungs It can also be expressed in terms of venous admixture

Clinical picture

History and progress

With rare exceptions there is a history of chronic bronchitis or asthma of several

years' duration, but the cardinal symptom of emphysema itself is excessive dyspnoea on exertion The onset is usually slow, but it may be abrupt, in which case there is commonly a history of antecedent acute respiratory infection Over a period of months or years the patient complains of increasing breathlessness, which may diminish from time to time with any improvement in the associated cough or broncho-spasm For this reason patients often state that they are better during the summer months

Cyanosis is usually inconspicuous, even in patients who are very dyspnoeic, but it may be severe, particularly in the terminal stages of the disease, when there may be secondary polycythaemia and clubbing of the fingers The primary cause of this cyanosis is inability to aerate the blood, but peripheral stasis and polycythaemia may also be important factors

The progress of the disease is usually insidious, but rarely it is rapid, and the patient may be incapacitated with severe dyspnoea within a year or two of the onset Usually there is a slow progression of symptoms over many years, and, if the patient does not die of intercurrent infection, the symptoms and signs of cor pulmonale and right sided heart failure may supervene The danger of right-heart failure is, however, unpredictable , some patients may be incapacitated by dyspnoea for years without any evidence of cardiac impairment, whereas others are first seen in heart failure, and will only admit a brief period of moderate dyspnoea The most common complication is said to be arterial hypertension Spontaneous pneumothorax, due to rupture of the flimsy visceral pleura may occur less frequent complications are haemoptysis and interstitial emphysema. Failure of vision due to papilloedema is very rare, but it occasionally occurs, usually in patients who have developed secondary polycythaemia

Physical signs

Although the significance of physical signs in the diagnosis of emphysema has been disputed, it is nevertheless true that in the advanced stages of the disease certain signs are usually present

Inspection.—The chest may be barrel shaped , thoracic kyphosis, loss of pulmonary elasticity and expiratory obstruction contribute to this deformity The relative importance of these factors varies from patient to patient, and it is not surprising that enlargement of the thoracic cage may be marked in some and inconspicuous in others, although they may be incapacitated by the disease If the enlargement of the thorax is considerable, the intercostal spaces are usually wide and the ribs ter [illegible] ...creased
The supraclavicular [illegible] ...hort
Expansion of the ch[illegible] ...neral
elevation of the thorax, heaving in character Expiration is often visibly prolonged

These phenomena are usually easily demonstrable, and they reflect the disorganization of breathing which accompanies loss of pulmonary elasticity and expansion of the thoracic cage The diaphragm, because it is no longer pulled upwards by the lungs, descends and becomes less convex, and its movement on inspiration is greatly diminished The intercostal muscles will also be unable to perform their proper function, because they are concerned with moderate inspiration and the chest is already expanded when inspiration begins , in order to expand

the chest further, the accessory muscles will have to be used, and these raise the front of the chest as a whole in a "heaving" manner, such as occurs in a normal individual towards the end of a forced inspiration Thus inspiration has to be performed by means of an unnatural inspiratory effort, and the patient with emphysema will encounter even greater difficulty in deflating his lung Normally, expiration is largely, if not wholly, a passive act, the thoracic cage is pulled inwards by the elastic recoil of the lung With loss of elasticity there must be loss of elastic recoil, so that if the lung is to be deflated it has to be squeezed The respiratory musculature was not built for this task, and the intercostal muscles have to be assisted by the accessory muscles of expiration The muscles of the abdominal wall can sometimes be felt to contract on expiration, which is prolonged as it is in other conditions, such as asthma and tracheal obstruction, in which the lungs are compressed by an active muscular effort With so extensive an impairment of both inspiration and expiration, it is not surprising that the vital capacity and chest expansion are reduced

The apical impulse is seldom visible or palpable, since a considerable thickness of inelastic lung is interposed between it and the chest wall In contrast, epigastric pulsation is often marked, presumably because of the low position of the diaphragm

Palpation—On palpation the vocal fremitus may be unaltered, but often it is diminished, possibly because bullae and enlarged air sacs are poor conductors of sound

Percussion—On percussion resonance is increased It is probably the superficial over distension of air sacs and formation of bullae which cause the remarkable hyper-resonance of the percussion note, which is sometimes asymetrical and which may be more resonant than over a pneumothorax The limits of lung resonance are increased because the size of the lung is increased, particularly in the region of its margins The potential gap between the chest wall and the heart becomes filled with emphysematous lung This space can be clearly seen in the lateral view by radiography, and it explains a variety of physical signs The absence of the apical impulse, loss of cardiac dullness, and distant heart sounds necessarily follow Similarly, the loss of liver dullness must in part be caused by expansion of the lung into the pleural sinuses, although the low position of the diaphragm is, of course, another factor

Auscultation—On auscultation the breath sounds are faint, particularly at the bases, but they may be increased in intensity by the concomitant bronchitis or broncho spasm, when coarse or sibilant rhonchi may be heard The factors leading to suppression of breath sounds are probably the same as those which cause diminution in the conduction of the spoken voice, but another theory has been proposed which may or may not play a part It is claimed that vesicular breathing in health is caused by the jet of air, which is thrown from the narrow alveolar duct into the wide atrium In emphysema, the alveolar duct is dilated so that this sudden change in calibre is lost, it is suggested that it is for this reason that vesicular breathing is faint or absent (Beitzke, 1925)

Expiration is prolonged, vocal resonance is usually diminished and the heart sounds are faint for reasons which are described above The second sound over the pulmonary area may be accentuated or reduplicated

Diagnosis

Interpretation of physical signs

The above are the physical signs of emphysema Although they are usually present in advanced cases, they may be absent in patients suffering from the disease and some are not uncommonly present in patients without emphysema Considerable confusion exists, even among physicians with wide clinical experience, as to the diagnostic value of these signs, and disagreement is perhaps greatest on the relationship of the barrel shaped chest to emphysema Some believe, and teach, that the physical signs associated with the barrel shaped chest, if definite, necessarily mean emphysema, although they admit that the lesion may neither progress nor lead to symptoms Others believe that the " barrel chest phenomenon " may occur quite independently of emphysema, and that in these circumstances it is of no significance Such a divergence of opinion as to the diagnostic criteria naturally makes for scepticism, and attempts to correlate the clinical diagnosis of emphysema with the facts as revealed by necropsy have led to further doubts It has been stated that only a small proportion of cases diagnosed at necropsy have been recognized during life and it has also been shown that many subjects with barrel shaped chests furnish no evidence of emphysema when they come to the post mortem table (Cabot, 1927 , Davidson, 1936 , Roelsen, 1938) For this reason some writers have gone so far as to say that the physical signs of emphysema are meaningless

Cabot states that of 12 cases diagnosed as emphysema at the Massachusetts General Hospital, only 3 showed emphysema *post mortem*, and that of 153 cases demonstrated *post mortem* only 7 had been recognized during life My own experience is rather different and is supported by a series of 72 cases diagnosed on the post-mortem table as generalized hypertrophic emphysema (Christie, 1944) All of these came from the medical wards of the London Hospital, but an accurate and detailed case-history was not always available, for some patients were moribund on admission A history of chronic cough was given in 68 cases and in a quarter of this number it was associated with mild or severe asthma Dyspnoea on exertion was present in 35 of the 45 case histories in which mention was made of the presence or absence of this symptom In the majority of cases, however, the physical signs of emphysema were not observed in 16 cases there was enough evidence to suggest an " emphysematous chest, but in only 6 cases could this evidence be said to be complete

This series of cases suggests that a more reliable guide to a diagnosis of emphysema than the physical signs is a history of chronic cough or asthma, associated with dyspnoea on exertion I have therefore investigated another series of 25 patients who gave such a history, and in whom no cause of dyspnoea other than emphysema could be discovered In 23 of them two or more of the physical signs

of " emphysematous chest "

no

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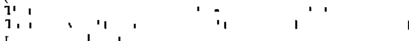
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In 9 of them the chest m ght have been said to be emphysematous and 4 presented the classical physical signs of emphysema

The conclusion that can be drawn is that the signs of emphysema are of significance but are unreliable they may be absent in patients suffering from the disease and are not uncommonly present in patients without emphysema A diagnosis of emphysema based only on physical signs may be fallacious since these depend largely on the barrel chest phenomenon which may occur independently of this disease In my opinion the diagnosis should only be cons dered to be definite when undue dyspnoea on exertion of insidious onset appears in a patient who has some of the physical signs of emphysema together with chronic bronchitis or asthma The absence either of dyspnoea or of physical signs should cast doubt on the diagnosis but should not exclude it the more physical signs present in a patient with chronic bronchitis or asthma the greater is the likelihood that he has emphysema I must confess however that in the absence of dyspnoea I am always uncertain as to diagnosis and prognosis but when dyspnoea not due to broncho spasm appears in a patient with chronic bronchitis I am very seldom in doubt A large series of cases will have to be followed up over many years before the necessary criteria for early diagnosis can be established Until this has been done I consider that the terms emphysematous and emphysematous chest should be discarded for it is their use which has been partly responsible for the lack of progress in the understanding of emphysema since its classical description over a century ago by Laennec These terms are quite misleading and should be replaced by some such expression as barrel chest or barrel chest phenomenon reserving the diagnosis of emphysema for those patients who have the symptoms as well as the signs of the disease I believe that if this were done the chances of progress would be increased particularly with regard to the use of special diagnostic procedures

Special d agnostic procedures

X ray examination —The appearances in emphysema are said to be characteristic



vertebrae The movements of the diaphragm on inspiration and expiration as well as those of the sternum are usually diminished The space between the sternum and the heart is almost invariably increased The heart may appear to lie vertically with a prominent pulmonary conus and exaggeration in size of the pulmonary arterial shadows at the lung hila The medium sized vessels tend to stand out more clearly but are smaller in calibre than normal the smaller peripheral vessels may also be more clearly visible than normal The lung fields may show increased translucency particularly at the lung bases Individual bullae may be seen usually at the apices of the lungs

It has been claimed that the radiological picture is a reliable guide to emphysema but attempts to establish the val dity of this claim have been unsuccessful In patients with florid emphysema a correct radiological diagnosis based on a postero-anterior film can b made in about 80 per cent of cases but if a control

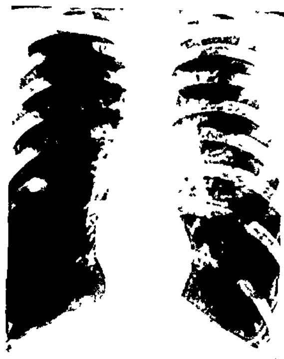


Fig 40—Radiograph of the chest in a case of chronic obstructive emphysema The intercostal spaces are wide and there is increased translucency of the lung fields especially at the bases The hilar shadow is prominent and the heart long and narrow The diaphragm is low and irregular in outline

group of elderly individuals be taken, about 20 per cent will be wrongly diagnosed as having emphysema A more reliable assessment may be made from films *taken in the antero-posterior and lateral positions, in both inspiration and ex-*

drawn from these observations is that the x-ray film may provide useful additional evidence , but taken alone it cannot exclude or establish a diagnosis of emphysema

Vital capacity and maximum breathing capacity —The vital capacity is usually reduced in emphysema Out of 30 patients with a clinical diagnosis of emphysema, I have found that in 25 cases the vital capacity was less than 70 per cent of the normal standard as calculated from the surface area, and in 15 cases it was less than 60 per cent In a control series of 30 patients of the same age distribution, without evidence of emphysema, the vital capacity was less than 70 per cent of the normal standard in 11, and less than 60 per cent in 3 cases There are many diseases of the lung other than emphysema which reduce the vital capacity, and it is clear that this test alone can be of little value in diagnosis

It is more easy to measure the chest expansion than the vital capacity, but unfortunately this also diminishes with age In my experience, about 60 per cent of patients with emphysema have a chest expansion of less than 1 inch , but I

have seen it reduced to ½ inch in a man aged 65 years in whom there was no other evidence to suggest pulmonary or cardiovascular disease

The maximal breathing capacity is also reduced in emphysema, but as a diagnostic test it suffers from the same drawbacks as does the vital capacity

Lung volume and its subdivisions —An important sequel to the dilatation of the air sacs in emphysema is the increase of the volume of the lungs as a whole which is usually obvious on the post mortem table the large fluffy lungs strongly suggest that during life there was an increase in the amount of air which they contained This amount can be measured, and early in the present century several investigators showed that there is usually an increase in the residual air in emphysema , but the residual air is the amount of air left after a forced expira tion, and it is well known that patients with emphysema may have great difficulty in forcibly emptying their lungs A more significant measurement is the amount of air in the lungs at the end of an ordinary quiet expiration—the functional residual air—for this is their true content during life, whereas the residual air is the result of respiratory gymnastics and may have no relation to the normal size of the lungs It has been shown that the quantity of the functional residual air is increased in emphysema, but this increase is not always proportionate to the severity of the disease In some patients it may be inconspicuous, when con trasted with the fairly wide range found in normal individuals In order to escape the need of normal standards which allow for body-size and other factors, it was

If a measurement is required to assist in the diagnosis or investigation of emphy sema this ratio is probably the most accurate measurement available , but it should not be forgotten that the ratio is quite an artificial figure and that any increase is due to two quite distinct factors , (1) the enlargement of the lung and (2) the inability of the patient to take a deep breath There has recently been a tendency to assume that an increase in this ratio, because it is a precise measure ment, is a more accurate diagnostic sign than is clinical evidence of the disease This has not been shown to be true

Tolerance to carbon dioxide —It has for long been known that patients with emphysema react sluggishly to the inhalation of carbon dioxide Although the fundamental cause of this impairment is unknown it is probably related to the high bicarbonate content of the blood and to inefficient ventilation in the lungs Recent improvements in technique allow the measurement of carbon dioxide tolerance to be made more easily than before, and it has been suggested that this reaction may be of value in the diagnosis of emphysema Even if the reliability of this test is established it is unlikely to become a routine diagnostic procedure, because special equipment is required (Donald and Christie, 1949)

The mixing of gases in the lung —In emphysema, the efficiency with which gases are mixed in the lung is grossly impaired (Cournand and his colleagues, 1941 Meneely and Kaltreider, 1949 , Bates and Christie, 1950) By means of the katharometer accurate measurements of the rate of gaseous mixing are now

possible, and it is likely that tests of this type will contribute to the problem of early diagnosis

Aetiology

Numerous hypotheses have been advanced to explain the cause of chronic hypertrophic or obstructive emphysema, but most are based on conjecture and are incapable of experimental proof or analysis Certain facts, however, stand out clearly almost all patients suffering from this disease are men of middle age or over who give clear evidence of chronic respiratory obstruction due to asthma or chronic bronchitis, or to persistent coughing from pulmonary tuberculosis, bronchiectasis, dusty occupations, or any other cause It has also been shown, both clinically and experimentally, that tracheal or bronchial obstruction, if sufficiently pronounced and prolonged, usually leads to emphysema This correlation between respiratory obstruction and emphysema cannot be denied, but the mechanism by which obstruction leads to the changes characteristic of emphysema is in considerable dispute

The expiratory theory

According to the expiratory theory, respiratory obstruction due to cough or bronchospasm will hinder exit of air from the lungs to a greater extent than entry, since the muscles of inspiration are more powerful than those of expiration As a result air will accumulate in the alveoli with subsequent distension and rupture of their walls This theory is simple and appears to be reasonable, but it does not take into account the immunity from emphysema of young persons who suffer from asthma The theory is also based partly on the false assumption that the lungs as a whole are grossly over distended It is true that the amount of air in the emphysematous lung is usually increased, but this increase is not always proportionate to the severity of the disease, nor is the volume of the emphysematous lung at rest as great as the volume of a normal lung in full inspiration , in most patients with emphysema the lung as a whole is not over distended any more than a normal lung is over distended when a deep breath is taken Incomplete emptying of the alveoli due to respiratory obstruction may be a factor in the production of emphysema, but it cannot be the only one

The inspiratory theory

This theory claims that over-distension is produced because too much air is drawn into the lungs by the increased inspiratory effort which precedes coughing, and which may be associated with asthma, with manual labour and with occupations such as glass-blowing and the blowing of wind instruments There is no evidence that these occupations or increased inspiratory effort due to any other cause play a part in the production of emphysema (Christie, 1939)

Impairment of pulmonary nutrition

[illegible]

in other diseases there may be more extensive [illegible]

emphysema although the lung does not become emphysematous For these reasons it is improbable that the vascular changes in the lung are of any aetiological significance

Degenerative or atrophic pulmonary char ge

It has been sugg ed ... or are secondary ... evade the issue s ... known to be inv ... explanations only ... count the many aetiological factors

Alveolar stress

Another theory is that the primary cause is increased stress and strain on the walls of the alveoli Almost all cases of emphysema have a long standing history of chronic bronchitis or asthma or both and this is also true of emphysema in horses and cattle The effects of cough and bronchial spasm on the lung as well as their mechanism are therefore of primary importance in any discussion on the aetiology of this disease

The mechanism of coughing is simple There is a short inspiratory effort which by no means fills the chest to full capacity the glottis is then closed and the lungs are forcibly compressed by all the muscles of expiration so that the pressure of air in the alveoli may rise to over 50 millimetres of mercury (Kountz and Alexander 1934 Rasmussen and Adams 1942) The glottis is then opened and the strain on the ribs the pressure on the pleura and the pressure in the alveoli are suddenly released In the case of the alveoli there must be a wave of pressure change passing from the bronchi out to the periphery of the lung

It is presumably this sudden release of pressure which is responsible for the sensation usually experienced that the lung is being distended but it is obvious from the mechanism of coughing that the lung is compressed and not in any sense distended This building up of pressure followed by its sudden release must mean increased stress and strain on all the structures involved especially on the alveolar walls which are not robust and which carry this wave of pressure change A chronic bronchitic patient may cough scores of times in a day and it seems reasonable to suppose that this type of stress and strain or wear and tear repeated over many years could in time produce the loss of elasticity and degenerative changes observed in the alveolar walls In many respects there is an analogy in arterial hypertension in which without any over stretching of the blood vessels there may be loss of elasticity and other degenerative changes and these are ascribed by recent investigators to the chronic stress of raised pressure It is also possible that wear and tear might occur outside the lungs when cough is chronic The pleura might suffer for the same reason as do the alveoli and the thoracic cage is also strained One has only to place a hand over the costal cartilages during coughing to realize that the impact is considerable In asthma there may be the same degree of change in intra alveolar pressure as in coughing and although the change is not so sudden it is repeated more often (von Neergaard and Wirz 1927 Hartwich 1930) This stress and strain repeated many thousands of times a day could have the same effect as chronic cough—loss of pulmonary elasticity The mechanism by which this loss may lead to many of the signs and symptoms of

emphysema is discussed above Loss of elasticity is probably an important factor in the production of emphysema, although resistance to expiration, as postulated in the "expiratory theory", may contribute to the progression of the disease

Almost all patients with emphysema suffer from either chronic bronchitis or asthma, and in these cases the explanation of the pathogenesis of emphysema which I have given is the most likely one It is, however, true that some patients with chronic bronchitis or asthma develop emphysema comparatively rapidly, whereas others never become emphysematous The forcefulness of the cough is obviously an important factor, and this *may vary greatly in patients suffering from* chronic bronchitis Clinical observation suggests that many patients cough in a *manner which is unlikely to strain the lung, but that others cough* with an impact which shakes the thorax There is, however, no clinical method of measuring the forcefulness of the cough, and the description given by the patient is usually quite unreliable Another factor is age, since with senescence there is a decrease in the resistance of the tissues to stress and strain This is particularly true of the *elasticity of the blood vessels With advancing years there may also be diminished* resistance in the lungs and in the elastic structures of the thoracic cage to the *stress and strain of cough or of asthma*

Emphysema without asthma or bronchitis

There still remains to be explained the unusual case of emphysema with no previous history of asthma or chronic bronchitis *I have seen 5 such patients in* whom shortness of breath preceded the onset of cough, and in one the diagnosis of emphysema was confirmed at necropsy An explanation, based on conjecture, that has been given is that these patients suffer from some inherent weakness of the elastic fibres in the lung It is also possible that, very rarely, the stress and strain on the lung which occurs with ordinary activities may be sufficient to destroy its elasticity

Hereditary and occupational factors

Other aetiological factors have been suggested, and a hereditary tendency to emphysema is one This may be so, but I have not seen it except indirectly as a possible familial tendency to asthma and chronic bronchitis *Hard manual labour* is another, but my own belief is that this is of importance only in that certain types of manual labour increase the incidence of chronic bronchitis It is a striking [illegible] scarcely a text book [illegible] nstru [illegible] ample evidence in the literature that no such aetiological [illegible] ristie, 1939) *The original statement of Laennec, made in 1819,* not based on any actual cases, has been copied from text-book to text book over a period of 120 years

Three probable factors

Such a variety of theories on *the aetiology of emphysema is confusing,* but the conclusion may be drawn that there are probably three factors of importance The first is inflation of the lung caused by obstruction to expiration The second

is loss of elasticity due to the stress and strain of coughing or asthma and a third is the lowered resistance of the lung to wear and tear which varies from person to person and which diminishes with advancing age

Treatment of chronic hypertrophic emphysema

The treatment of emphysema is essentially symptomatic for elastic tissue cannot regenerate and nothing can restore the structure of the lungs Until heart failure supervenes the only symptom other than cough is dyspnoea on exertion Therefore I first emphasize the value of broncho dilator drugs

Broncho dilator drugs —Although there may be no evidence of bronchospasm or resistance to respiration the administration of ephedrine or of other broncho dilator drugs not infrequently relieves the dyspnoea of emphysema A possible explanation of this effect is that the bronchioles leading to the over distended air sacs and bullae are less capable of changes in calibre than are those leading to healthier parts of the lung bronchospasm although not clinically manifest, might in this case increase the proportion of the inspired air deflected to these useless parts of the lung and the relief of bronchospasm by ephedrine medication would improve the efficiency of ventilation and thus relieve dyspnoea The broncho dilator drugs which may be used and their method of administration are similar to those recommended in the more chronic phases of bronchial asthma (*see* p 92)

Treatment of bronchitis —Bronchitis should be treated in the usual way (*see* Volume I p 58) but the results are usually disappointing except when contributory factors such as dusty occupations or smoking can be prevented The response to antibiotics is variable but is seldom impressive

Pulmonary deflation —Several procedures the purpose of which is to deflate the lung have been described and it is said that these increase the efficiency of res piration Pneumothorax is one and relief of dyspnoea by this procedure both in human beings and in horses suffering from this disease has been claimed Pneumothorax in emphysema however is a hazardous procedure More popular in Great Britain and less dangerous are respiratory exercises designed to promote relaxation and to teach the patient to deflate the lung and to increase the use of the diaphragm A well fitting abdominal belt will also raise the diaphragm by increasing intra abdominal pressure and more recently pneumoperitoneum has been suggested for the same purpose All these precedures have been recom mended and various theories have been put forward to explain the beneficial effects observed Two effects are common to most of them First the diaphragm is raised so that its convexity is increased and its efficiency therefore enhanced and it was for this reason that I have suggested the use of the abdominal belt Secondly the lung is deflated by these procedures so that it contains less air The decrease in volume is however very small—so small that it can hardly be measured—and the beneficial effects are probably for the most part due to increased efficiency of the respiratory musculature It is also possible that with greater collapse of the superficial bullae efficiency of ventilation may be increased

Treatment of heart failure —When heart failure supervenes the general principles recommended in the treatment of cor pulmonale should be adopted Oxygen should be given because the added insult of anoxia to a heart that is failing

greatly lessens the chances of recovery but it should be given with care for the sudden relief of anoxia may lead to convulsions and coma It is believed by some physicians that digitalis should be avoided particularly in an emergency Venesection has of course been recommended but it is now seldom used Mercurial diuretics may however be of considerable value Heart failure is often precipitated by respiratory infection commonly subacute broncho pneumonia and this usually responds to treatment with antibiotics It is because broncho pneumonia is no longer a lethal complication that recovery from heart failure in emphysema is now a common occurrence

Conclusions —The treatment of emphysema is thus far from being hopeless although it is essentially symptomatic *Bronchitis should be treated and in all* cases broncho dilator drugs should be given a trial Respiratory exercises or an abdominal belt or both may increase considerably the tolerance to exercise



Fig 41 —*A case of senile emphysema* The wizened appearance the upper thoracic kyphosis the bulging of the sternum and the relative increase in the antero posterior diameter are characteristic There is no increase in the volume of the thoracic cage

SENILE EMPHYSEMA

Synonyms Atrophic emphysema postural emphysema arteriosclerotic emphysema non obstructive emphysema small lunged emphysema senile atrophy of the lungs

Clinical picture

As its name implies this is essentially a disease of old age (*see* Fig 41) The patient often presents a shrivelled and withered appearance The chest is usually barrel shaped but there is no significant enlargement of the thoracic cage The *bony changes may be more pronounced* than in the obstructive type of emphysema

and n the l ter stages

the

The appearance of the lung is variable *In some cases the proc* [illegible] ects the thoracic cage in others there is chronic bronchitis and this complicates the picture *by introducing the obstructive element* Occasionally this atrophic process is believed to affect the lung itself but this has not been established On the *available evidence* no clear description of the lung in senile emphysema can be given it may be normal or it may show the characteristics found in the obstructive

type of emphysema, but the lungs are usually small and bullae are seldom conspicuous

The symptoms and signs of senile emphysema bear little relationship to the apparent deformity Symptoms are often absent, even in the presence of extreme kyphosis More rarely there may be all the symptoms and signs characteristic of chronic obstructive emphysema

Aetiology

This condition is probably but one of the atrophic changes occurring in old age Changes in the thoracic cage may be ascribed to atrophy of the intervertebral discs, leading to kyphosis, with consequent rotation of the ribs, so that the chest tends to become barrel shaped Atrophy of the alveolar walls is said to lead to their disintegration but there is often an associated chronic bronchitis which exaggerates the emphysematous changes

Diagnosis

Differentiation from obstructive emphysema —Senile emphysema differs from the chronic obstructive form in the following respects It is a disease of old age rather than of middle age The cough is seldom severe and dyspnoea and cyanosis are usually absent The kyphosis of the upper thoracic vertebrae is characteristic Although barrel shaped, the chest is not enlarged Chest expansion may be impaired, but usually inspiration has not the same heaving quality as in the obstructive type

Treatment

Treatment, if any is indicated is similar to that of chronic obstructive emphysema

ACUTE VESICULAR EMPHYSEMA

This is an ill defined entity , it includes several conditions in which transient signs of emphysema appear

For instance, during an acute attack of bronchial asthma the increased expiratory resistance may lead to distension of the lung, which subsides when the attack is over Similarly, in acute respiratory infections, diphtheria, whooping cough or any condition associated with transient respiratory obstruction, the lungs may become large This temporary distension does not rupture the alveolar walls nor, in fact does it lead to any permanent damage but, as stated above, it may be a contributory factor to the production of obstructive emphysema

Even less well defined are the changes sometimes observed after congestion of the lungs Hyper resonance of the lung has been described following acute heart failure, strenuous exercise or septicaemia , it has also been found in the contra lateral lung in pneumothorax and massive pleural effusion, and after residence at high altitudes To interpret this change as being indicative of emphysema does not appear to be justified

LOCALIZED OR FOCAL EMPHYSEMA

Synonyms Compensatory emphysema , complementary emphysema , vicarious emphysema

Pathogenesis

fails to expand on inspiration, either the surrounding alveoli must become distended to occupy the space, or the chest wall must be drawn in Both phenomena usually occur if the contraction is sufficient to displace the mediastinum, and signs of emphysema may appear in the contralateral healthy lung

Focal emphysema in pneumoconiosis —In most cases this form of emphysema is a pathological finding rather than a clinical entity, but an important exception is in pneumoconiosis Recent investigations have shown that the emphysema which occurs in silicosis differs from obstructive or hypertrophic emphysema in its focal distribution Around the pneumoconiotic nodules the air spaces may be dilated to a diameter of several millimetres, and these foci of emphysema are widely scattered throughout the lung, particularly in the secondary lobules (Gough, 1940 and 1947) The clinical picture is, however, much the same as in chronic obstructive or hypertrophic emphysema, with cough, dyspnoea and rarely cyanosis, and ultimately evidence of cor pulmonale The aetiology of this type of focal emphysema remains obscure The relative importance of cough and of contraction due to fibrosis is unknown , it is possible that the condition is one of ordinary obstructive emphysema, but the morbid appearances are modified by fibrosis and scarring, and by the widespread pleural adhesions which are common

Lobar obstructive emphysema —Another type of localized emphysema, often lobar in distribution, may be a sequel to partial obstruction of a bronchus in conditions such as carcinoma of the lung and tuberculous bronchitis If there is sufficient obstruction to the flow of air through the bronchus the elastic recoil of the lung may be inadequate to deflate the alveoli beyond the obstruction and inflation of this part of the lung will result This condition has been called lobar obstructive emphysema, and if the obstruction is of long standing, it is claimed that gross over distension with bulla formation may result Sometimes the obstruction leads to a "ball valve" occlusion of the bronchus which allows the entry of air, but not the exit, and under these circumstances inflation of the lobe may be considerable

ACUTE INTERSTITIAL EMPHYSEMA

Interstitial emphysema, or passage of air into the connective-tissue spaces, is usually a sequel to injuries of the chest or lung , it may appear after tracheotomy It is a rare complication of severe asthma, when the cause is presumably rupture of the lung itself The air may escape into the interlobular and sub-pleural spaces, or it may be extravasated along the walls of the bronchi and blood vessels to the mediastinum, passing up to the subcutaneous tissues of the neck and face

There are not usually any symptoms, and the condition is first suspected when the characteristic crepitus is discovered in the subcutaneous tissues of the neck. Sometimes there are cyanosis and alarming dyspnoea, and on auscultation peculiar

crunching and crackling sounds may be heard over the heart Recovery is the rule and very rarely, if ever, is operative intervention indicated to relieve air which is under tension (Rosenberg and Rosenberg, 1938 , Wallace, 1946 , d Abreu, 1948)

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CHAPTER 10

PULMONARY OEDEMA

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PHYSIOLOGICAL FACTORS

THE PHYSIOLOGICAL factors responsible for the formation of oedema fluid in the lung are fundamentally the same as those concerned in the production of oedema elsewhere in the body, modified in some respects owing to differences in the pulmonary and systemic circulation. The factors to be considered are (1) hydrostatic pressure relationships, (2) osmotic factors, and (3) permeability of the capillaries.

Hydrostatic pressure relationships

Direct measurement of the pulmonary arterial pressure in the unanaesthetized human by means of cardiac catheterization is now possible, and it has been found that the systolic pressure in the pulmonary artery is between 15–30 millimetres of mercury (Cournand, 1947). Direct measurement of the pulmonary venous pressure in a normal man is not possible but catheterization of the pulmonary veins in patients with an atrial septal defect has given pressure readings of 5–7 millimetres of mercury (Cournand and his colleagues, 1947). The pressure at the arterial end of the pulmonary capillary is about 10 millimetres of mercury, as compared with a pressure of 25–30 millimetres in a systemic capillary. The pressure in the interstitial tissues of the lung is approximately that of the atmosphere so that the effective filtration pressure tending to cause transudation of fluid through the capillary membrane is also about 10 millimetres.

Osmotic factors

The osmotic pressure of the blood in the pulmonary vessels is the same as that elsewhere in the body, that is 25–30 millimetres of mercury with the normal plasma protein level of 7 grammes per cent. This is the force which tends to prevent leakage of fluid through the capillary wall.

Permeability of the capillary membrane

It is customary to imagine that the capillary wall acts as a semi-permeable membrane, allowing crystalloids to pass readily in either direction, but retaining protein in the vessel. Studies of the lymphatic flow in animals show that usually a small amount of protein and fluid escapes from the vessel into the interstitial tissue of the lung and is rem[illegible] the lymphatics. In conditions in which the permeability of the pulmonary capillary is increased, plasma may escape in large amounts and the clinical condition of pulmonary oedema results. The permeability of these capillaries may be affected by a variety of factors, some humoral, such as anoxia, others neurogenic, such as trauma to the heart or brain, and others toxic.

In an[illegible]

pulmonary capillaries would be accompanied by increased permeability and the formation of oedema fluid

Lymphatic drainage

The lung is freely supplied with lymphatics which in health are easily able to remove the small quantities of plasma which escape from the capillaries Changes in lymphatic flow when the permeability of the lung capillaries is increased have been studied by Drinker (1945) As a result of anoxia, or the administration of phosgene or thiourea compounds, the lymphatic flow is considerably increased As more fluid escapes from the vessel, the lymphatics may be unable to remove all of the fluid, which then escapes into the alveoli causing pulmonary oedema It is probable that a similar sequence takes place in man

From these physiological considerations it may be seen that in the normal person the relatively low filtration pressure and high osmotic pressure will reduce the transudation of fluid to a minimum, and any fluid which may escape is efficiently removed by the lymphatics If, however, the permeability of the capillary wall is increased, either through the influence of anoxia, toxic substances, or nervous stimulation, or if in addition the pulmonary capillary pressure is increased by either an increase in the pulmonary arterial pressure or pulmonary venous pressure, fluid may pass into the interstitial tissue and alveoli of the lung more quickly than it can be removed by the lymphatics, and the condition of pulmonary oedema will result

CLASSIFICATION

Pulmonary oedema occurs in such a wide range of clinical conditions that an aetiological classification is unsatisfactory A clinical classification, based primarily on the type of pulmonary oedema is probably to be preferred

(1) Acute pulmonary oedema
- (*a*) Associated with heart failure (left ventricular failure, coronary thrombosis, acute cor pulmonale)
- (*b*) Associated with damage to or diseases of, the central nervous system (head trauma cerebral vascular accidents space occupying lesions of the brain)
- (*c*) Following lung irritants such as phosgene

(2) Subacute
- (*a*) Associated with left ventricular strain
  - (i) rheumatic pneumonia
  - (ii) uraemia
  - (iii) polyarteritis nodosa
- (*b*) Disturbances of fluid balance
  - (i) desoxycorticosterone acetate poisoning
  - (ii) transfusion
  - (iii) stilboestrol

(c) Associated with lung irritants
Infections (pneumonia, virus infections, industrial irritants)

(3) Chronic
(a) Associated with heart failure—hypostatic congestion
(b) Associated with lung irritants—chronic infections

It would be redundant to describe each of these types in detail since much of what applies to one, applies to the others The attention of clinicians and experimentalists has been mainly focused on acute pulmonary oedema associated with heart failure, and for this reason discussion of the fundamental principles involved will be for the most part confined to this section

ACUTE PULMONARY OEDEMA

Associated with heart failure

The syndrome of acute pulmonary oedema associated with heart failure has also been called cardiac asthma, or paroxysmal nocturnal cardiac dyspnoea These are all manifestations of acute pulmonary congestion any of which may develop into acute pulmonary oedema In the cases labelled "cardiac asthma" there is in addition a varying degree of bronchospasm, and the term paroxysmal nocturnal cardiac dyspnoea merely indicates two of the well recognized clinical features of the attack It would be better to adopt the term acute pulmonary congestion, with or without pulmonary oedema, for all cases

The first clinical description of this condition was made about two centuries ago, and clinicians from the time of Laennec have favoured the simple hypothesis that the cause is acute failure of the left ventricle to expel the blood delivered to it by the right ventricle with consequent engorgement of the pulmonary vascular bed This purely mechanical concept of the pathogenesis of acute pulmonary oedema has, however, little to support it either clinically or experimentally

The basis of the mechanical theory of blood accumulating behind a failing left ventricle lies in the experiments of Welch in 1878 on rabbits He interfered with the function of the left ventricle by crushing it or by ligation of the aorta and produced pulmonary oedema His results are still quoted, but have been criticized on the grounds that the appearance of pulmonary oedema was inconstant, spontaneous pulmonary oedema frequently occurs in rabbits, the nature of the experiments resulted in gross damage to the animals, and finally that it has been impossible to reproduce the results in other animals Numerous experiments designed to change mechanically the circulation in the lungs by constriction of the pulmonary veins have shown that it is difficult to produce pulmonary oedema in this way Experiments with the heart lung preparation have been quoted in support of the theory of left ventricular failure, but it is now agreed that the oedema produced was usually due to chemical changes in the perfused blood

In place of the mechanical theory of the cause of pulmonary oedema, there has been a tendency among physiologists to state that pulmonary oedema is caused by neurogenic vasodilatation of the pulmonary capillaries and resulting increased permeability The stimulus may arise from direct excitation of nervous centres, as in head injuries, or reflexly from stimuli arising in the heart or large vessels (Luisada, 1940)

The main conclusion that can be drawn from the mass of experimental evidence in the literature is that although pulmonary oedema can be produced by various mechanical and reflex procedures, the only reliable method of producing acute pulmonary oedema in animals is by means of substances which act directly on the pulmonary vascular bed, such as anoxia, phosgene and certain other chemicals, notably the thiourea derivatives It should not be forgotten that it has so far proved impossible to produce chronic pulmonary congestion or in fact any condition in animals that resembles the clinical picture which is known to predispose to acute pulmonary oedema or which resembles the actual clinical attack of acute pulmonary oedema

The vast majority of patients with acute pulmonary oedema give a history of chronic left ventricular failure with dyspnoea and orthopnoea, and in these the incidence of attacks is greatly reduced when right heart failure supervenes This suggests that pulmonary congestion may at least play an important part, and this view is strengthened by the clinical observation that venesection or the application of limb tourniquets which reduce the degree of pulmonary congestion are useful procedures in the treatment of an acute attack

The experimental and clinical evidence is not, however, wholly contradictory Although acute pulmonary oedema occurs most commonly in patients with chronic left ventricular failure and pulmonary congestion it may also follow cerebral accidents or right ventricular strain such as occurs in pulmonary infarction, acute cor pulmonale, pneumonia and thrombosis of the coronary vessels supplying the right ventricle In this group there is nothing to suggest left ventricular failure and yet frank pulmonary oedema may occur It is possible that in these cases reflex vasodilatation and increased permeability of the pulmonary vessels is the cause, the stimulus arising from the heart, great vessels, or the brain

The clinical observation that the attacks usually occur during sleep has been the source of much speculation Increased autonomic activity when the patient is awakened by dreams or coughing may lead to pulmonary vasodilatation, or, in patients who are orthopnoeic, slipping down in bed may lead to increased pulmonary congestion Another explanation which has some evidence to support it is that a sudden movement while asleep may cause a considerable increase in the venous return to the heart (Christie, 1938) Even in the healthy individual a transitory increase in the heart rate of 30–40 beats a minute may occur during sleep, and with the venous stagnation which occurs in heart failure it seems reasonable to suppose that, after a period of relaxation, a sudden movement during sleep might increase considerably the influx of blood to the heart

The conclusion which can be drawn from this conflicting evidence is that we do not know the essential cause of acute pulmonary oedema, but that certain contributory factors appear to be of importance A background of pulmonary congestion favours the onset, and the trigger mechanism which initiates the attack must be increased capillary permeability due either to reflex vasodilatation or to a sudden increase in the return of venous blood to the heart The reflex stimulus may arise in the heart itself, from a myocardial infarct or sudden pressure changes in any of the heart cavities or great vessels , it may arise from the lungs,

as in pulmonary infarction or lung irritants or from the higher centres from emotion excitement or trauma

Aetiology

In heart failure acute pulmonary oedema is most commonly associated with hypertension aortic valvular disease particularly aortic regurgitation and coronary artery disease It may also occur as mentioned above in right heart failure such as occurs in pulmonary or myocardial infarction In all of these pulmonary congestion is a prominent feature Attacks of acute pulmonary oedema may occur in patients with tight mitral stenosis In these patients who often give a history of recurrent haemoptyses there is little enlargement of the heart or left auricle the pulmonary arteries are prominent and the pulmonary artery pressure is raised Acute pulmonary oedema is rarely seen with auricular fibrillation

Pathology

The pathological changes in the lung are those of widespread oedema both interstitial intra alveolar and filling the larger air passages so that on section the cut surface exudes frothy fluid The lymphatics and pulmonary capillaries are widely dilated As seen at autopsy the whole of both lungs are affected The changes in the heart are those of the underlying disease

Clinical course

Acute pulmonary oedema associated with heart failure usually occurs in patients giving a history of increasing cardiac dyspnoea and orthopnoea With little or no warning and usually at night he suddenly develops intense and terrifying dyspnoea He sits up or tries to get out of bed struggling for breath Coughing at the onset is frequent and aggravates the dyspnoea and the onset of bronchospasm may further embarrass his breathing Substernal tightness is often experienced together with a feeling of suffocation The attack may stop almost as suddenly as it started or it may increase in severity to a condition of gross pulmonary oedema The cough becomes more frequent and frothy or watery sputum sometimes blood stained may be brought up

When seen during the attack the patient appears in obvious respiratory distress Sweating and anxiety are marked Cyanosis appears early and if the attack continues will become marked Examination of the lungs in the early stage of the attack may show little or no abnormality Some rales are usually heard first only in the mid zone of both lungs and later extending and becoming coarser until the breath sounds may be obscured by the added sounds The heart rhythm is nearly always normal sinus rhythm and as Bedford (1939) has pointed out it is unusual to see attacks of acute pulmonary oedema in patients with established auricular fibrillation In the latter condition both ventricles are commonly failing and it is well recognized clinically that with the onset of right heart failure attacks of acute pulmonary oedema may cease There is evidence of left ventricular hypertrophy in most cases the notable exceptions being those patients who have coronary heart disease and myocardial infarction without an associated hypertension The pulmonary second sound is accentuated and there is frequently

a presystolic triple rhythm—'gallop rhythm'—at the apex Except in those cases due to myocardial infarction, the blood pressure during an attack is raised or unchanged until the terminal stage of an attack, when it may fall A significant degree of *pulsus alternans* may often be detected when the blood pressure is being measured

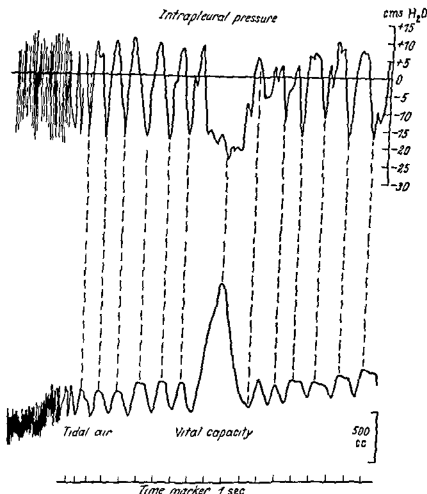


FIG 42—Simultaneous recordings of intrapleural pressure and tidal air in a patient with heart failure showing the increased rigidity of the lung caused by pulmonary congestion The pleural pressure fluctuation is 27 centimetres of water (10 to −17) Normal fluctuation 2 7 centimetres of water (*From Christie and Meakins 1934*)

Intense dyspnoea dominates the picture this is not due to the stimulus of carbon dioxide retention or of anoxia but is a direct result of pulmonary congestion (Christie, 1938) The lungs become more rigid and less distensible (Fig 42) and reflex dyspnoea follows through stimulation either of the Hering

Breuer reflex or of vagal nerve endings in the pulmonary vascular bed (Bulbring and Whitteridge, 1945)

Cyanosis, since it can be relieved by oxygen, must be for the most part pulmonary in origin and is due to the exudate in the alveoli which impedes gaseous exchange. The elimination of carbon dioxide is not significantly impaired, partly because its dissociation curve differs from that of oxygen and partly because it is more soluble and can diffuse more readily through the alveolar exudate

Bronchospasm is variable in its intensity and is usually ascribed to congestion of the bronchial mucosa through the anastomotic channels which connect the bronchial and pulmonary vessels This can hardly be called an explanation since the bronchospasm may be relieved by adrenaline and is presumably autonomic in origin.

Why the attack passes off is even more uncertain than why it should start It may be that a rapidly failing left ventricle and increasing anoxia in the tissues leads to generalized vasodilatation Blood would thus be transferred from the pulmonary to the systemic vascular bed, with consequent rel[illegible] congestion That this does indeed happen [illegible], that the termination of these [illegible] [illegible]ans, the pulse is often imperceptible, [illegible] change to a dusky grey

The electrocardiogram, befor[illegible] during an attack, will often show the changes caused by marked left ventricular hypertrophy The limb leads show left axis deviation with flattening or inversion of T_1 The praecordial leads, V_1 and V_2 (over right ventricle) show a small R and deep S, whereas in V_5–V_7 the R wave is tall and the T wave inverted

Radiological findings

Radiological changes in the lungs accompany the appearance of clinical signs of oedema in the lungs The typical early appearance is that of symmetrical areas of increased density spreading out from both hila to give a butterfly wing appearance (Fig 43) At this stage, the periphery of the lung fields and particularly the lung bases may appear unduly clear Careful examination of the areas of increased density will show a diffuse haziness obliterating the lung markings, unlike the changes in chronic congestion where the lung markings may be increased and the dilated hilar vessels clearly seen If the attack is short lived these changes disappear quickly, and all radiological signs may have disappeared in 4–6 hours If the attack progresses to massive pulmonary oedema, the areas of haziness will spread to involve all of both lungs (Roesler, 1943)

Other investigations

No accurate measurements of cardiac output or pulmonary arterial pressure during an attack are available The circulation time is prolonged and the vital capacity diminished A marked degree of arterial anoxaemia will be found during the attack, and cyanosis may be exaggerated by the haemoconcentration caused by the loss of a large amount of protein-containing fluid from the circulation

Treatment

An attack of acute pulmonary oedema is a medical emergency, and prompt



(a)

FIG 43—(a) Skiagram during an attack of acute pulmonary oedema in a patient with hypertensive heart disease (b) skiagram 12 hours later showing considerable resolution



(b)

treatment in the early stages may abort the attack, whereas if delayed until the later stages, treatment may be difficult

Oxygen—The relief of anoxia is important as the pulmonary capillaries themselves may under the influence of anoxia become more permeable with resultant increasing pulmonary oedema and more anoxia Prompt administration of oxygen will help to break this vicious circle It should be given either by B L B mask, or if this is not tolerated, by spectacle frame nasal catheters A flow of 5–8 litres per minute is usually recommended, even if the more efficient B L B mask is used Because of restlessness and the feeling of suffocation, an oxygen tent is rarely tolerated

Sedatives—Sedatives are necessary in this condition to relieve the patient's distress during the attacks They may also be helpful because if, as is possible, a nervous reflex mechanism is responsible for initiating the attack, the sedative by depressing reflex action may help to stop it The drug of choice is morphine which should be given subcutaneously in doses of $\frac{1}{4}$–$\frac{1}{2}$ grain, or intravenously in those patients with circulatory collapse Alternatively, the barbiturates may be used, particularly the rapidly acting ones

Procedures acting probably by lowering the venous pressure—Rapid venesection of 600 millilitres of blood in those patients with a considerable increase in venous pressure will often help to terminate the attack The use of tourniquets around the thighs, to obstruct venous return, has also been advised for the same purpose Apart from morphine probably the most useful drug in the treatment of an attack is theophylline with ethylenediamine In addition to causing a fall in venous and right auricular pressure (Howarth, McMichael and Sharpey Schafer, 1947), it will also relieve bronchospasm It should be given intravenously in a dose of 0 24–0 48 gramme diluted in 10 millilitres of distilled water The value of atropine is disputed It may help to dry up an excess of bronchial gland secretion, but its effect on the pulmonary nerve endings in man is unknown The place of [illegible]
[illegible]
[illegible]
dosage to control the ventricular rate

Prevention of attacks—Any procedure which reduces pulmonary congestion [illegible]

Prognosis

The occurrence of attacks of acute pulmonary oedema in a patient with heart disease is a sign of serious prognostic importance The average duration of life after the first attack in a series of patients with left ventricular failure was only 2 years (Bedford, 1939)

Pulmonary oedema associated with lesions of the central nervous system

Severe injuries to the skull or brain not infrequently cause acute pulmonary oedema, and Weisman (1939) found that 70 per cent of patients dying after a fractured skull had signs of oedema of the lungs It occurs less frequently in

diseases of the central nervous system such as cerebral haemorrhage, and occasionally is associated with brain abscess or tumour

This type of pulmonary oedema which occurs in patients with no disease of the heart, or history of chronic pulmonary congestion, is of particular interest to the experimental physiologist, as it resembles closely the type of acute pulmonary oedema produced in animals by a wide range of experimental procedures The cause is presumably increased permeability of the pulmonary capillaries, without the added factors of left ventricular failure, and therefore without massive pulmonary congestion The cause of the increased capillary permeability which follows damage to the brain is unknown, but it is presumably due to reflex dilatation of the pulmonary capillaries and consequent increase in permeability

The clinical picture of a patient with this type of acute pulmonary oedema differs from that seen in left ventricular failure Dyspnoea does not dominate the picture—probably because of the absence of gross pulmonary congestion The respirations are often deep, laboured, and stertorous The frequent absence of orthopnoea is difficult to evaluate because of the disordered consciousness of the patient More prominent in this traumatic type of oedema are moist sounds in the lung, with cyanosis, and cough with thin expectoration which may later become frothy It is usually a terminal condition, and fails to respond to the methods of treatment which have been outlined in the previous section

SUBACUTE PULMONARY OEDEMA

Associated with severe hypertension

An unusual type of pulmonary oedema which is subacute in its clinical course is seen in several diseases, all of which are associated with left ventricular strain

The changes in the lungs in rheumatic pneumonia as described by Hadfield (1938) consist of an initial fibrinous alveolitis with later infiltration of the exudate with mononuclear cells and eventual organization when fibroblasts may be seen The alveolar ducts are lined by a hyaline membrane The lungs macroscopically are solid or rubbery, particularly in the central position near the hilum Other changes due to the rheumatic infection are seen, and in all cases carditis is present, often with a fibrinous pericarditis These changes in the lung were thought by many to represent the specific pulmonary lesion of the rheumatic infection

The clinical picture of a patient with the 'rheumatic lung' is initially that of a severe rheumatic carditis, with later increasing dyspnoea and often slight cough. The physical signs in the lungs are inconspicuous compared with the extent of the lesions seen radiologically Slight impairment of percussion note and breath sounds in the mid zone with a few fine crepitations may be detected but not infrequently the lungs show no abnormality on physical examination The chest skiagram shows a characteristic change, with central cloudy opacities, often confluent, extending peripherally into the lung with the lung bases and apices remaining clear (Fig 44)

An exactly similar condition is seen in uraemia (Doniach, 1947), in acute nephritis and in polyarteritis nodosa, when there is severe hypertension It is [illegible] The radiological and histo- [illegible] with those seen



FIG 44 — Subacute pulmonary oedema in rheumatic fever (autopsy control)



FIG 45 — Subacute pulmonary oedema in malignant hypertension (autopsy control)

It is probable that in all of these diseases the same pathological process is taking place, and that the clinical, radiological and pathological findings are caused by increased capillary permeability, usually in association with left ventricular failure with resulting pulmonary oedema, both interstitial and intra alveolar (Epstein and Greenspan, 1941) The difference between this subacute pulmonary oedema and the acute type described above, presumably lies in the fact that in the former the exudate clots and then becomes organized, with cellular infiltration In acute pulmonary oedema the exudate is removed before clotting and organization has time to occur It is of interest that the same histological appearance in the lungs can be produced in animals by various procedures which cause pulmonary oedema, such as the administrations of large doses of thiourea derivatives and in sulphonamide hypersensitivity

Subacute pulmonary oedema caused by a disturbance of fluid balance

Intravenous therapy

In patients with a normal cardiovascular system it is difficult to produce circulatory overloading, even when comparatively large amounts of fluid or blood are given The increased venous return to the right heart increases the output of the heart, and the additional fluid is accommodated by the opening up of reserve capillaries in the lungs and elsewhere in the body In patients who are ill, particularly those with anaemia associated with chronic infection, quite small amounts of blood or saline solution given intravenously may cause circulatory overloading and a subacute type of pulmonary oedema may be produced In such a patient, the earliest signs will be a
engorged neck veins a slight rise in resp
dilatation Auscultation of the lungs at
but if the transfusion is continued, moist sounds will appear at the lung bases and frank pulmonary oedema may result The x ray findings are increased prominence of the vascular markings at the lung roots with generalized loss of translucency of the lung fields most marked at the lung bases

nary oedema is presumably
The myocardium in the
mplication of intravenous
therapy shows the fatty change due to anaemia and toxaemia, and failure occurs when the heart is unable to compensate for the increased venous return and increased circulatory blood volume

The treatment of this condition is both prophylactic and curative If the danger of producing pulmonary oedema in patients with chronic anaemia and sepsis is recognized, transfusion should be given slowly, and the use of packed cells instead of whole blood to reduce the bulk of fluid given is advised If pulmonary oedema does occur during a transfusion, the transfusion should be stopped, possibly temporarily and if gross oedema is present, the immediate administration of a mercurial diuretic, such as mersalyl or Neptal is advisable The marked diuresis which follows will help to remove the excess of fluid from the body and the pulmonary oedema may disappear

Administration of desoxycorticosterone acetate (D O C A) and stilboestrol

The synthetic suprarenal cortical hormone D O C A differs from the naturally occurring hormone in that a more marked retention of sodium and water occurs during the period of administration of the drug If given to normal healthy persons, a gain in weight due to the increase in extracellular fluid occurs (Thorn and Emerson, 1940) The same applies to patients with Addison's disease who are given excessive doses of D O C A , and when the sodium intake in the diet is not restricted, pulmonary oedema may be produced This type of oedema is not associated with marked pulmonary congestion, so that although the respiratory rate may rise, dyspnoea is often not marked Râles may be heard over both lungs, and the chest skiagram shows the typical appearance of diffuse pulmonary oedema The condition when recognized usually subsides rapidly when D O C A. is discontinued

Retention of sodium is also known to occur as a result of the administration of stilboestrol and pulmonary oedema has occasionally been noticed during treatment of the metastases of a prostatic carcinoma. The correction of anaemia by transfusion in patients under treatment with either of these drugs should be carried out with caution as this may precipitate an attack of pulmonary oedema

Subacute pulmonary oedema associated with lung irritants

A subacute type of pulmonary oedema is seen in several infections of the lung, notably in the virus infections causing primary atypical pneumonia The clinical features of this disease are described elsewhere Radiologically, the soft patchy opacities often centrally placed, resemble the changes described above as typical of subacute pulmonary oedema, and in the few cases which have come to autopsy a similar histological appearance has been noted (Jenson, 1946 , Golden, 1944)

Industrial irritants are described in Volume I, Chapter 6

CHRONIC PULMONARY OEDEMA

Hypostatic congestion

In patients with chronic left ventricular failure, a degree of persistent oedema of the lung bases is frequently seen A similar condition may occur in elderly patients who are confined to bed Presumably as a result of impaired diaphragmatic movement, slowing of the circulation and anoxia there is increased permeability of the capillaries and oedema at the bases Treatment is that of the underlying condition and early mobilization of the patient

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CHAPTER II

ATELECTASIS

F P LEE LANDER

THE WORD "atelectasis" means "imperfect expansion" (*Oxford English Dictionary*) It was first used in the English literature in 1846, in Swaine's translation of Hasse's *Pathological Anatomy* Shortly afterwards Rees (1850) wrote a short monograph entitled *Atelektasis Pulmonum* in which he gave the following definition

> Imperfect expansion Atelektasis is intended to designate a condition of the lung in which there is simple occlusion or shutting up of the pulmonary air cells without any inflammatory or other morbid deposits

Since that time the term has been in current use, and though a few writers have regarded it as describing collapse of congenital origin only, the majority have not so limited its use The morbid anatomists of the middle nineteenth century were fully aware of the frequent occurrence of the condition, and advanced many theories as to its formation

Legendre and Bailly (1844) identified broncho-pneumonia as a form of atelectasis, and produced some convincing experimental work to support their theory

Gairdner (1851), in his treatise on bronchitis, mentioned the frequency with which the condition occurs, and stated that it was the commonest complication of any bronchitic condition Furthermore he produced, for the first time, the theory of the mucus plug, and drew a diagram indicating the way in which the plug would be affected by changes of pressure

From the literature it appears that the attention of the morbid anatomists was then distracted from the condition, for in the latter part of the nineteenth century little reference was made to it, and it was left to William Pasteur (1908), in his Bradshaw Lecture, to re-discover it and to connect it with diphtheria and later with post-operative complications Pasteur distinguished two varieties massive collapse due to diaphragmatic paralysis, which he called active collapse, and lobular collapse due to bronchial obstruction, which he called passive collapse

An answer to many of the questions posed by Pasteur, and a negation of most of his theory is offered by the extensive use since his time of the operation of diaphragmatic paralysis by phrenic nerve interruption This diaphragmatic paralysis, we know from experience, is not necessarily or even commonly attended by massive collapse, and it seems probable that in the cases described by Pasteur diaphragmatic paralysis was attended by obstruction of the finer bronchi, although it is possible that in some of his cases elevation of the diaphragm and restricted movement secondary to the massive collapse were mistaken for actual paralysis of the diaphragm Nevertheless, Pasteur's work remains a classic, largely because he described, for the first time, the detailed physical signs and attributed them rightly to lessened movement of the diaphragm

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and no other compensatory mechanism may be called into play As the child grows, and the thorax enlarges, then the collapse becomes manifest with the development of the other compensatory mechanisms

Gairdner, in 1850, drew three simple diagrams to explain his theory of the production of emphysema In these diagrams, Fig 46*a*, showing three equal partitions represents three lobes or portions of a lung Fig 46*b*, demonstrates that when one portion of the lung has collapsed the other two portions expand by drawing in air, to fill the whole space Fig 46*c* shows that when two portions

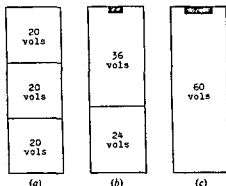


FIG 46 — Diagrammatic representation of

space

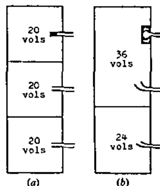


FIG 47 —(*a*) The same as in Fig 46(*a*) but including bronchi (*b*) expansion of bronchus when a lobe collapses

of the lung collapse the remaining portion expands to fill the whole space In Gairdner s original diagrams, he showed an equal expansion of the two remaining portions of the lung As a result of study of radiographs and lobectomy specimens it is now known that the expansion is not evenly distributed through the thorax, but is more marked nearer the collapsed portion

So far we have been considering the behaviour of the alveoli When considering the behaviour of the bronchi exactly the same considerations apply The bronchi are highly elastic tubes the part of the bronchus distal to the obstruction (that is cut off from the outside air) will shrink because it is in unobstructed communication with the alveoli, and replenishment of air cannot take place The part of the bronchus proximal to the obstruction being in free communication with the outside air will therefore expand (Fig 47)

The walls of the thoracic cage are also elastic and expansile and consequently will be affected by any diminution of the volume of any part of the thoracic contents The area involved will be that which is closest to the shrinking volume of the lung The mediastinum is readily mobile and displacement of the heart occurs in atelectasis involving the lower parts of the lung on either side, this

Today, whilst bronchial obstruction is regarded as the most important, if not the only, method of producing atelectasis, a great deal of confusion still exists as to the actual mechanism by which it is produced Furthermore, the compensating mechanisms which are called into play by the shrinkage of the atelectatic area are only just beginning to be studied and understood

PHYSIOLOGY

When a bronchus is completely obstructed, alveolar air is absorbed and cannot be renewed This absorption of air is attended by the shrinkage of the lung and this keeps the pressure of air in the obstructed alveoli constant If there were no shrinkage, pressure of air would fall and absorption of air would cease when the alveolar air pressure was at the same level as the partial pressure of the gases in the venous blood The shrinkage is rendered possible by the elasticity of the alveolar walls, which not only allows the obstructed lung to shrink, but also allows the unobstructed lung to expand and fill the space left by the retracting obstructed lung

This space is filled also by contraction of the walls of the thorax, by a move of the mediastinal structures towards the retracting lung, and also by an elevation of the diaphragm on the affected side All these factors can be seen and verified, clinically and radiologically They do not all occur in each case to the same degree in some cases the unobstructed lung expands to such an extent that no other mechanism is necessary to fill the space A further effect of shrinkage is a compensation by the bronchi in the unobstructed lung, which dilate to beyond their normal range

The size of the thorax varies with respiration, and consequently the degree of compensation required is greater in inspiration, when the thoracic volume is at its maximum, than in expiration This can be watched on the fluoroscopic screen, and demonstrated by inspiratory and expiratory radiograms Clinically, however, a diminution in the respiratory movements of the affected side can be seen, thus a limitation in the increase in thoracic volume on the affected side is one of the compensatory mechanisms

Occasionally, in cases of complete bronchial obstruction, as air absorption occurs secretion is poured into the bronchi and thus the gaseous pressure in the obstruction system is maintained at a high level and this continues until all air is absorbed and the state then exists of an airless lung with complete bronchial obstruction and no shrinkage

All states between the condition just described, through a range of reduced secretion and increased shrinkage, to a completely dry atelectasis, can occur

If the main bronchial obstruction persists absorption of this secretion may occur, with gradual shrinkage of the obstructed portion of the lung

In the case of children, a slightly different state exists because the child s thoracic volume is small as compared with the adult's thoracic volume, and the child's lung increases in volume enormously as growth occurs and adult stature is attained The increase in size occurs largely by an increase in size of each individual alveolus Consequently, when atelectasis occurs in a child's lung, compensation takes place simply by enlargement of the adjacent alveoli,

In certain cases a new growth of a main bronchus may not, by itself, produce complete obstruction The bronchial lumen however, may be narrowed so much that the obstruction is made complete by thick, tenacious mucus As a result, air is absorbed in the usual way behind the obstruction and the mucus is sucked down the bronchial tubes If a mass of mucus is small, an airway may be re established and the condition of the lung return to normal (Fig 48) However, if the mucus is of sufficient volume the obstruction may be complete (Fig 49)

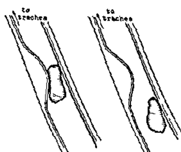


FIG 48—(a) Small mass of mucus in bronchus, (b) airway re-established

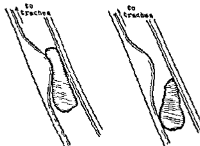


FIG 49—(a) Large mass of mucus in bronchus (b) the obstruction is complete

This mucus plug will be sucked down the bronchus splitting up at each division until finally the original plug is split into a thousand fragments obstructing a thousand terminal bronchi In this way lobar collapse may be produced by

complicating cases of tuberculous tracheo bronchitis

Obstruction of finer divisions of the bronchi leads to segmental atelectasis and obstruction of terminal bronchi leads to lobular atelectasis. The nature of the obstruction in the finer bronchi is not certain as direct observation is impossible Spasm of the bronchial muscle, oedema of the bronchial mucous membrane, and fine plugs of mucus probably all play a part These fine plugs of mucus have been demonstrated in autopsy specimens in post operative cases Whooping-cough, asthma and bronchitis are commonly complicated by this degree of atelectasis, and tuberculous tracheo bronchitis is a less common cause

SYMPTOMS

Massive atelectasis is usually attended by a dramatic onset Sudden urgent dyspnoea, pain in the chest and palpitations are frequent Occasionally cyanosis also occurs

Lobar collapse is usually less obtrusive in its onset A pain in the chest or tightness may be noticed dyspnoea is sometimes present, and palpitations may occur

displacement being usually accentuated in inspiration The trachea is displaced by atelectasis of the upper lobe on either side, and the pulmonary arteries are also pulled up and distorted

Atelectasis of one complete lung produces the maximal effect on the thoracic boundaries It has been shown that the atelectatic lung lies in the paravertebral sulcus behind the hilum, the anterior part of the affected side being occupied by the heart and by the lung from the other side The diaphragm is involved in all these cases of lobar collapse In complete collapse of one lung, elevation of the diaphragm is extreme, and the diaphragm is immobile In lower lobe collapse elevation and flattening of the diaphragm occurs, with restriction of the diaphragmatic movement In upper lobe collapse, elevation may not be so marked, but restriction of movement occurs In lobular or segmental collapse the effects are more limited, and distortion of the mediastinum occurs as well as localized effects on the diaphragm

CAUSATION

Any obstruction of a bronchus, if complete both in the inspiratory and expiratory positions, is attended by atelectasis of the corresponding bronchi and alveoli The obstruction may be situated anywhere between the epiglottis and the finest contributory bronchus When obstruction of the trachea takes place, death from suffocation occurs before the lungs are completely airless When obstruction of one main bronchus takes place, atelectasis of the corresponding whole lung occurs When obstruction of a primary division of a main bronchus takes place, collapse of one lobe of the lung occurs, and so on, until finally obstruction of one terminal bronchus leads to collapse of a small group of alveoli

Atelectasis may be classified, according to the extent of lung involvement, as (a) massive (one whole lung) and lobar atelectasis and (b) lobular and segmental atelectasis

This classification is convenient as it corresponds roughly to the different aetiological causes Atelectasis of a whole lung is usually seen in the condition of carcinoma of the bronchus Occasionally post operative atelectasis may also involve the whole of one lung Inhalation of a foreign body may also produce this condition Trauma to the chest, especially crush injuries, may sever a main bronchus and produce a traumatic stricture leading to a permanent atelectasis of that part of the lung In childhood the adherence of a tuberculous gland to a main bronchus, with erosion of the wall of the bronchus and the production of an intense oedema of the bronchial mucous membrane, can cause complete obstruction and consequently collapse of a whole lung This condition may also occur in adult life Occasionally obstruction of a main bronchus by glandular enlargement due to one of the reticuloses may produce the condition

The above-mentioned conditions may produce blockage of a primary lobar bronchus, and consequently lobar collapse Obstructions in the main bronchi or their primary branches are readily visible through the bronchoscope

Tuberculous tracheo-bronchitis sometimes produces obstruction, either of a main bronchus leading to massive atelectasis, or of a primary (lobar) bronchus leading to lobular atelectasis

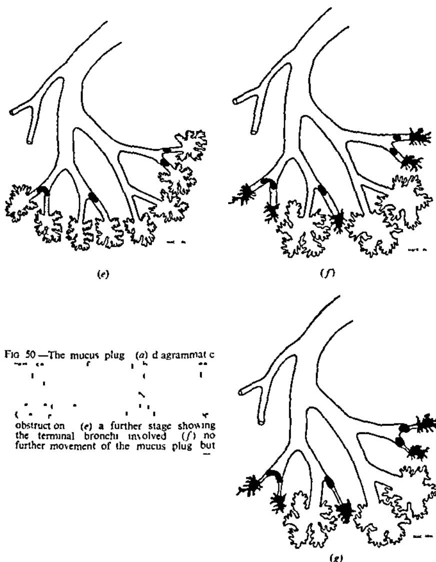
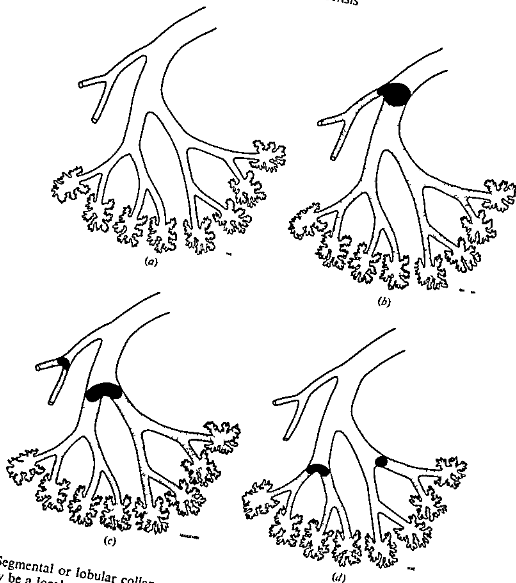


FIG 50—The mucus plug (a) diagrammatic [illegible]

obstruction (e) a further stage showing the terminal bronchi involved (f) no further movement of the mucus plug but

the affected lung elevation of the diaphragm with displacement of the gastric hyper resonance up into the axilla may lessen the area of dullness and lead to some confusion The air entry is variable In the early stage there may be complete absence of breath sounds especially in cases of main bronchial obstruction such as new growth or inhaled foreign body In other cases loud breath


(a) (b) (c) (d)

Segmental or lobular collapse usually passes unnoticed and the only symptom may be a localized pain over the area

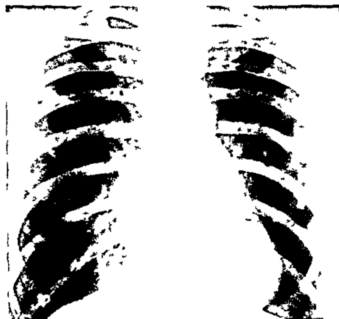
PHYSICAL SIGNS

When a whole lung is atelectatic the signs are well marked Flattening and immobility of the affected side of the chest are present The trachea is deviated towards the affected side and the apex beat is widely displaced towards the same side The percussion note is dull over the affected lung though if the left is



FIG 51 —(*a*) Atelectasis of left lower lobe The heart is slightly displaced to the left and there is an area of emphysema in left costo phrenic angle The shadow of the collapsed lobe cannot be seen (*b*) crowding together of the lower lobe bronchi with a small amount of dilatation and the lingula bronchi which have come down to occupy the space left by collapsing lobe

(*a*)



(*b*)

sounds of a tubular character may be present from the beginning. Added sounds are not usually present.

In lobar collapse, similar signs to a lesser degree may be present. Thus, the trachea and heart are rarely both displaced. In upper lobe collapse, the trachea is displaced towards the affected side and the heart is not disturbed, the opposite being the case in lower lobe collapse when the heart is displaced towards the affected side and the trachea remains central. Dullness is present over the collapsed lobe which in the case of the lower lobe lies against the spine posteriorly when the area of dullness is triangular in shape and does not extend further forward than the mid axilla. In the case of the upper lobe, the dullness is confined to the anterior part of the upper chest, and is not usually extended below the third rib. The signs produced by collapse of the middle lobe are so slight that they are difficult to elicit. Dullness over the front of the chest, from the fourth rib downwards, may be present, extending to the anterior axillary line. The breath sounds over the collapsed lobe are usually absent, but may be tubular and loud.

RADIOLOGICAL SIGNS

The radiological signs are characteristic, but vary with the duration of the collapse. In early cases, a homogeneous opacity of characteristic shape (the character varying with the affected part of the lung) will be seen. This opacity is usually attended by the signs of the compensatory mechanisms already outlined—a localized area of compensatory emphysema, deviation of the heart and trachea, and elevation of the whole or part of one diaphragm.

In adult cases, one or all of these radiological abnormalities will be present. In children, especially in cases in which one lobe or less is involved, the compensatory mechanisms may not be present and may only be established if the atelectasis persists with the growth in stature of the child.

INVESTIGATION AND TREATMENT

In massive or lobar collapse, the possibility of new growth of the lung or foreign body should always be borne in mind and bronchoscopy undertaken. The possibility of tuberculosis should also be investigated by examination of several specimens of sputum for tubercle bacilli. In the more extensive cases, this possibility may be confirmed by the finding of ulcerative lesions in the bronchi.

When the diagnosis is made within a short time of the onset of symptoms then postural drainage with percussion over the affected part of the chest may dislodge the foreign body or mucus plug. In cases of lesser degree an antispasmodic, such as ephedrine, ½ grain by mouth or tincture of stramonium, 15 minims, is worth administering and repeating at 4 hourly intervals. Penicillin should be given to all patients in whom there are signs of infection in the atelectatic lung. The intramuscular route is the method of choice, as the intrabronchial obstruction may prevent the penicillin reaching the area infected if it is administered by inhalation.

COMPLICATIONS

Infection

If virulent organisms are trapped behind a bronchial obstruction, an acute febrile

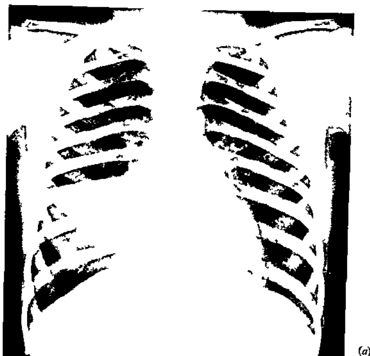


(*a*)



(*b*)

FIG 53 —(*a*) Atelectasis of whole of left lung The trachea is not apparently deviated but no heart shadow is seen to the right of sternum (*b*) complete filling of all bronchi with very marked increase in size of left main bronchus and also lower part of trachea (*c*) the whole lung is crowded together into a small area against the posterior chest wall



(a)



(b)

FIG 52 (a) Collapsed m ddle lobe The e s a sharp upper demarcat ng l ne ta l ng off into normal lung mark ngs in the lower lobe There s no sh ft of the med ast num (b) the typ cal shadow of a col lapsed m ddle lobe



(a)



(b)

F G 54 —(a) Comple e a e ectas s of left lung w h r ght lung be ng pu ed o er and fi l ng up part of the space left by the reced ng lung (b) the whole of he a electa c lung s ly ng aga nst pos e or chest wa



FIG 53 (c)

illness is likely to be set up resembling lobar pneumonia and leading possibly to break-down of lung tissue with the formation of a lung abscess In cases with a less virulent infection the febrile illness will be less dramatic in its onset and course

Rupture of an emphysematous bulla

In a number of cases the compensating emphysema which occurs is so localized and so marked that rupture of one of the bullae occurs and the condition of spontaneous pneumothorax is produced This complication is one that occurs at the same time as the atelectasis and persists after the lung has re aerated

POST OPERATIVE ATELECTASIS OF THE LUNG

This variety of atelectasis is particularly favourable for study and treatment It occurs most frequently after operations on the mouth nose and throat but also complicates all types of operations The variety of anaesthetic used does not influence its incidence to any great extent but intubation of the larynx appears to increase the frequency of its occurrence

Atelectasis occurs within 48 hours of the operation and may have a dramatic onset resembling that of a lobar pneumonia

The patient may have a sudden pain in the chest with breathlessness and a rapid rise of temperature up to 104°F With this type of onset it is usual to find large



(a)



(b)

FIG 54.—(a) Complete atelectasis of left lung with right lung being pulled over and filling up part of the space left by the receding lung (b) the whole of the atelectatic lung is lying against posterior chest wall



F g 55 — Atelecta c r ght lower lobe w th heart and med ast num pulled across towards the same s de

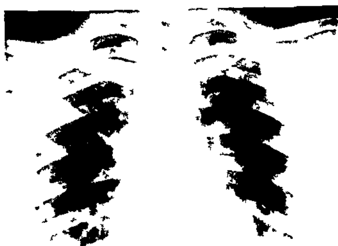
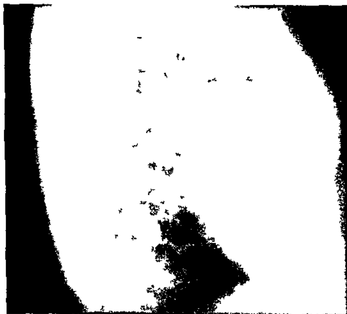


Fig 56 — (*a*) Honey comb ng in upper zone of r ght lung The trachea s central w th no apparent de viat on of med asti num (*b*) bronch of upper lobe are crowded together (*c*) complete fill ng of upper lobe wh ch occup es a very much smaller area than normally with marked crowd ng together of bronch and with saccular d latat on



(b)



(c)



(a)

Fig 57 — (a) Opacity due to a complete obstruction of left upper lobe bronchus with consolidation of upper lobe and little shrinkage (b) and (c) bronchograms showing the extent of the upper lobe Bronchoscopy in this case showed *the obstruction to be complete* and due to a carcinoma of the *bronchus (d) air has been* induced into pleura showing *a marked shrinkage* of upper lobe



(b)



(c)



(d)

areas of lung involved and consequently physical signs are found Dullness to percussion over the affected area with deviation of the cardiac impulse (and possibly the trachea) to the affected side are found The auscultatory sounds are variable At first there is a complete silence over the dull area but later loud bronchial breathing may be heard and later still numerous moist sounds.

It is more usual to have a quieter onset, in which a low fever is noted on the temperature chart, and on questioning the patient admits to a slight cough dating from the operation The physical signs also are inconspicuous but definite, small areas, at the bases of lungs of moist sounds are heard with bronchial breathing in the same areas

The time incidence of the temperature coupled with the physical signs in the chest establish the diagnosis of atelectasis

The treatment depends on the extent of the lesion and the earliness of the diagnosis

If a large area of lung is involved and the condition is recognized within 24 hours of the onset then bronchoscopy should be performed and attempts made to aspirate them

With smaller areas or a later recognition this procedure is valueless, as the mucus is then in the finer bronchi, out of range of the bronchoscope

Postural drainage should be used and vigorous percussion over the affected area of lung applied in an attempt to dislodge fine plugs and enable the patient to cough the plugs out

Discretion in the use of these methods is necessary, frail old age after extensive resection operations being treated more gently than robust youth after an appendicectomy

Prophylaxis plays an important part in the treatment of this condition Preoperative breathing exercises are valuable but the chief measure is to avoid an operation in the presence of an upper respiratory infection or bronchitis

In operations on the upper respiratory passage attention to bleeding points and the disposal of small pieces of tonsil, teeth and mucous membranes is of great importance

Frequent change of posture of the semiconscious patient immediately after operation and for the next 24 hours can also help to minimize the effects of mucus in the air passages

THE FATE OF THE ATELECTATIC LUNG

For how long a lobe can remain atelectatic and then become re aerated is a matter of considerable speculation When the obstruction is permanent, such as that caused by a carcinoma of the bronchus, a bronchial stricture secondary to a tuberculous process, or an undisturbed foreign body, the collapse is permanent When the obstruction is temporary, such as that caused by a plug of mucus or oedema of the bronchial mucous membrane, then the atelectasis is as temporary as is the obstruction, and may last for any period from a few minutes to a few weeks In post-operative cases, in which it is possible to assess the moment of onset, clearing has frequently been found to be complete in 1 week, but has also persisted for 2–3 weeks, and yet ended in complete re-aeration of the affected lung In cases which persist for longer than 3 weeks, it is safe to say that

although some re-aeration may occur, large areas of atelectasis are likely to persist

The majority of cases do re expand completely, yet such is the frequency of atelectasis that many patients are seen in whom the atelectasis has persisted, often only recognized when one of the late results calls attention to the condition

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CHAPTER 12

BRONCHIECTASIS

F P LEE LANDER

HISTORY

THE WORD "bronchiectasis" first appeared in the English literature in 1846, when the Sydenham Society published Swaine's translation of Hasse's *Pathological Anatomy* The word is derived from two Greek words—*bronchos* meaning a tube, and *ectasia* to stretch

In 1821 Laennec had described for the first time a condition of dilatation of the bronchial tubes, his attention having been drawn to the condition by his pupil Andral who had noted it at post-mortem examination Laennec's description in the first edition of his great work *Mediate Auscultation* was brief, but in the fourth edition the description was enlarged to include the clinical findings of cases which later came to post-mortem examination He described the condition as occurring in the upper lobes of the lungs, and the clinical differentiation from pulmonary tuberculosis was made mainly on the length of the history and on the appearance of one or two added sounds, to which today we attach no importance He did not describe clubbing of the fingers as occurring in the condition, nor did he lay any stress on displacement of the heart and trachea His theory of the pathogenesis was that the dilatation of the bronchial tubes was caused by a copious secretion of sputum distending the bronchi, and the copious secretion of sputum he considered to be the primary disease

Andral (1831) mentioned the condition, but did not advance any theory as to causation, though he did remark that while generally the dilatation occurred in those who had suffered from chronic cough for a great many years, it also occurred as a complication of whooping cough in children who, previous to the whooping cough, had had no cough at all

Stokes (1837) drew a parallel between aneurysm of the aorta and dilatation of the bronchi He suggested that the elastic tissue in the non muscular part of the bronchial wall was destroyed, that there was a paralysis of the circular muscles due to inflammation, and that the cilia lost their power of movement These three causes, he considered, contributed to the dilatation of the bronchial tubes and the stasis of their contents

The early theorists regarded the disease as primarily one of the bronchial wall, and considered that the changes occurring in the surrounding lung tissue were secondary to the bronchial change The first person to challenge this concept was Corrigan, who, in 1836, maintained that the lung changes were primary and that as a result of the contraction in the lung, bronchial dilatation was produced His theory was that a fibrosis occurred in the lung, secondary to some chronic disease, and that the contraction of this fibrous tissue produced dilatation of the bronchial tubes

Hasse subscribed to the theory that bronchial dilatation was part of a compensating mechanism, secondary to the collapse of the lung, and stated that bronchitis was one of the major causes.

On the other hand, Gairdner, in 1850, came to the conclusion that the so-called bronchial dilatation was in reality ulcerative excavation of the lung, which became relined by bronchial mucous membrane.

Since that time the majority of theories advanced have been in favour of the primary disease being one of the bronchial wall, and much of the research has been directed into proving this theory, showing that disease in the bronchial wall results in the destruction of the elastic tissue, so that as a result of the increased pressure, either of a forced inspiration following a cough or because of the raised intratracheal pressure due to the forced expiration against the closed glottis, a dilatation of the bronchi is produced which becomes permanent. Of recent years, however, there has been a swing in thought to the ideas put forward by Corrigan (1836), the only difference being that whereas he postulated a fibrosis or "cirrhosis" of the lungs secondary to chronic infection, the current theory is that the shrinkage of the lung occurs largely as an acute process following peripheral bronchial obstruction and atelectasis.

Until 1922 the actual proof of the presence of the dilatation was found only at post mortem examination. In 1922, however, Sicard and Forestier introduced the use of opaque oil in radiography for the study of the hollow viscera of the body, thus enabling bronchiectasis to be diagnosed, with certainty, in life. The immediate result of this outstanding contribution to medical science was the stimulation of interest in bronchiectasis and a consequent outpouring of articles. Amongst these was one by Wall and Hoyle (1933), in which the concept of the condition was enlarged from the confines of the heavily infected extensive cases to the almost symptomless cases of dilatation.

Articles by Finlay (1935) and Warner (1934) drew attention to the association between dilatation of the bronchi and collapse of the lobes of the lung.

Smith (1930) demonstrated the appearance of spirochaetes in the sputum of bronchiectatic patients, and attributed the bronchiectasis to this organism. Since then however, the presence of spirochaetes has been demonstrated, times without number, in the sputum of bronchitic patients, and those suffering from every type of respiratory disease, with or without bronchial dilatation.

Jennings (1937) published a case in which well marked dilatation of the bronchi was shown later to have disappeared and the bronchi to have returned to normal. Since then many such cases have been demonstrated, and the name of "reversible bronchiectasis" has been given to the condition. Some writers state that this condition has nothing to do with permanent bronchiectasis and consequently have labelled it "pseudo-bronchiectasis". Modern American writers in discussing the reversible changes in the bronchi, state that the appearances of the dilatation in a collapsed lobe are possibly due to a shortening of the bronchi, which, at the same time, produces a widening of the calibre. Against this theory is the fact demonstrated by Macklin (1929) that with expiration the bronchi narrow and shorten. Shortening of the bronchi is not necessarily attended by dilatation. There are cases on record in which collapse of the lung is attended

by shortening of the bronchi with no alteration in calibre. This can be demonstrated also in cases of artificial pneumothorax.

Andrus (1937) examined in detail all the physical forces in the lung occurring with various conditions, and came to the conclusion that the forces arising as a result of pulmonary atelectasis provide the most satisfactory explanation of the disease.

EXPERIMENTAL WORK

Tannenberg and Pinner (1942), in their experiments, produced a main bronchial obstruction in dogs, and the final state of the bronchi seen at post-mortem examination was found to depend on the presence or absence of infection. Where infection has been added the bronchi were found to be dilated, whereas where no infection had been added the bronchi were collapsed and the walls touching.

Adams and Escudero (1938) also did a series of experiments on dogs, but they were only able to produce bronchiectasis when the obstruction was incomplete and when contamination of the bronchial tree had occurred. In none of their cases with complete obstruction, even in the presence of infection, were they able to demonstrate bronchial dilatation. The demonstration of its presence or absence was again made at post-mortem examination.

Weinberg (1937) produced bronchial obstruction in rabbits by means of a sand burr. Every possible attempt was made to exclude infection, and although this was not successful in each case, bronchiectasis was the universal finding, also demonstrated at post-mortem examination.

Davidson and Lee Lander (1938) produced bronchiectasis in cats by the introduction of gum acacia. In this case the demonstration of the dilatation was made by means of a bronchogram in the living animal.

These varying, and apparently contradictory results must be made to fit into some all-embracing theory of the production of the dilatation. In interpreting any experimental work the first essential is to note in each case whether the bronchiectasis was demonstrated by bronchography in life or at post mortem examination. This is an important point because, if modern theories are correct, the dilatation is, in the first place, dependent on the normal physical conditions existing in the intact thorax, and removal of the lungs from the thorax *post mortem* destroys these physical conditions. Corrigan (1836) was the first to point out the importance of the rigid walls of the thorax for the production of the condition. This was also clearly illustrated in one case seen at the Brompton Hospital in the early days of lobectomy, when a lobe was removed from the thorax for the condition of bronchiectasis and subsequent examination of the lobe showed it to be dense and airless but with completely normal bronchi. The pre operative bronchograms in this case clearly illustrated the presence of a fusiform bronchiectasis.

The second point to note is the position of the obstruction, either central or peripheral. All air spaces distal to the obstruction are subject to the same forces, whether the obstruction is central or peripheral, and similarly conditions proximal to the obstruction are identical; thus bronchi peripheral to the obstruction will collapse. The main difference is that in the case of a central obstruction the bronchial walls are strengthened by cartilaginous rings and are consequently

less easily distensible or collapsible. Once this point is realized a great many of the apparently divergent results in the experimental work will fall into line.

The third point to note is the extent of the obstruction in the main bronchus. If the obstruction is incomplete, collapse will not follow unless at the site of the narrowing of the main bronchus mucus forms and renders the obstruction complete.

The theory first propounded by Gairdner (1851), that the bronchial dilatations are produced by an actual destruction of the bronchial wall, with a complete loss of all the normal structures of the wall and the resulting cavity then becoming relined by bronchial mucous membrane still has some adherents (Lisa and Rosenblatt, 1943). In support of this theory the histological appearances of cystic dilatations have been quoted. The point that is overlooked is that the structure of the bronchial wall is not uniform throughout its length. In the region of the lung hilum the bronchial wall is a complicated structure, containing glands, muscle, elastic tissue, fibrous tissue, and cartilage, whereas in the periphery of the lung it is simple and consists of bronchial mucous membrane and a thin layer of muscle and elastic tissue. Consequently, the interpretation of the histological appearances of any cavity associated with the bronchus must be correlated with the position of the cavity in its relation to the bronchus.

The possibility of a congenital origin for bronchiectasis has been raised, and there is a certain group of cases still referred to as congenital cystic bronchiectasis. This theory is an extremely difficult one either to prove or discount. The frequent association between bronchiectasis and other congenital abnormalities, such as transposition of viscera (Adams and Churchill, 1937) or cystic conditions of the pancreas (Anderson, 1938; Baylin, 1944), kidney, and liver, have been used as an argument in favour of the congenital origin. Some writers have suggested that congenital bronchiectasis arises as a maldevelopment or non-development of parts of the primitive lung bud. Others have suggested that obstruction of the bronchial tree occurs during intra-uterine life, probably with amniotic fluid, and that this obstruction prevents expansion of parts of the lung. Neither of these possibilities can be completely ruled out in any case occurring within the first few weeks of life. Many of the cases showing the so-called typical appearance of congenital cystic lung can be shown to occur in children whose lungs have been known previously to be healthy. The proof of the presence or absence of a developmental abnormality must rest finally on the demonstration of cystic spaces in the still-born lung.

It has already been mentioned that peripheral bronchial obstruction is the important factor in the production of bronchiectasis. Whether the patient suffers from the clinical syndrome of long-standing cough, foul sputum, and haemoptyses depends on the second factor, which is the presence of infection.

AETIOLOGY

Patchy atelectasis occurs in many conditions in childhood. The two most frequent causes of this condition are whooping-cough and measles. These two diseases consequently are the commonest causes of bronchiectasis. Enlarged

tuberculous glands in the hilar region of the lungs may produce lobar collapse and account for a number of cases of bronchiectasis (Brock, Cann and Dickinson, 1937) Brock (1950) drew attention to this as a cause of haemorrhagic bronchiectasis of the right middle lobe.

Inhaled foreign bodies are responsible for a small number of cases, and post-operative atelectasis, more especially following operations in the upper respiratory tract, account for a further number Later in life there are many cases of atelectasis of obscure origin, which may persist and give rise to permanent collapse and bronchiectasis Later still carcinoma of the bronchus plays an increasing part in the production of the condition

Lobar pneumonia has always been ascribed an important role in the aetiology of bronchiectasis, and in cases in which resolution is delayed or incomplete, bronchiectasis does follow These cases of unresolved pneumonia are probably due to retention of secretion in the consolidated lung leading to a permanent atelectasis It has been stated that the occurrence of bronchiectasis in cases with asthma is rare, and that atelectasis also does not occur in spite of the bronchial oedema and spasm A thorough search by bronchography in cases of asthma does not bear out this statement, both atelectasis and bronchiectasis being frequent complications of asthma

Many children with bronchiectasis give a history of a tracheotomy during an attack of diphtheria and a tracheotomy scar is found on examination of the neck There may be no connexion between these two facts Pasteur, however, pointed out that atelectasis was a frequent occurrence in severe cases of diphtheria It is also possible that the tracheotomy was performed on the mistaken diagnosis of laryngeal obstruction, the obstruction in fact being lower down the respiratory passages

Case report

J T a small boy aged 3 years was seized with intense dyspnoea and cyanosis Marked stridor was present and a tracheotomy was performed as an emergency measure No relief was obtained and the child was admitted to hospital On admission dyspnoea and cyanosis were present and on examination of his chest complete dullness and absent air entry on the left side were found

Bronchoscopy was performed His larynx and trachea were found to be normal The mucous membrane of the left main bronchus was oedematous and intensely inflamed and the oedema completely obstructed the lumen of the bronchus The right main bronchus was normal No cause for this was found The left lung remained massively collapsed and bronchiectasis was demonstrated in the collapsed lung (*see* Fig 58)

Many other factors have been put forward as the cause of bronchiectasis Infection of the nasal sinuses has been ascribed a major role, and the frequency with which the two conditions coexist has been used as a proof of their relationship It has been shown that inspiration of opaque oil into the nose at night has produced widespread dissemination of the oil in the lungs the following day This shows that it is possible for mucus dropping down the back of the nose to be inspired, and possibly produce obstruction and collapse

I G 58 — (*a*) Mass ve bronch ectas s of left lower lobe 1936 Few bronch on the r ght outl ned are w1 h n normal l m ts (*b*) 1946 comple e collapse of left lung w h bronch ectasis n olv ng all bronch on left s de bronch of right m ddle lobe have already come acros the m dl ne of the left thorax but a l bronch outl ned are normal



(*a*)

(*b*)

Fibrosis of the lung is not a primary condition, and probably plays no part in the production of bronchiectasis In cases of silicosis the widespread fine fibrosis is an essential part of the disease, and bronchography has demonstrated normal bronchi in the *majority of cases*

Bronchial dilatation is a frequent complication of tuberculous conditions of the lung, more especially in those cases in which atelectasis has played a major role, and in conditions of tuberculous tracheobronchitis, bronchiectasis is an invariable accompaniment

The histology of bronchiectasis, obtained from post mortem specimens, has nearly always shown the presence of gross infection, and this fact has been used to give infection a dominating role in the production of the condition Histology of lobectomy specimens on the other hand, when the lobectomy has been performed in early cases or cases in which there were few symptoms, has shown little, if any, infection

Bacteriology of the sputum is extremely varied It is unusual to find one organism as the only inhabitant of bronchiectatic sputum, and it is much more usual to find 5 or 6 varieties of organisms, none of them predominating The bacteriology closely resembles that of bronchitis

DIAGNOSIS

Symptoms and signs

[illegible] on the presence of dilated bronchi but on infection [illegible], if virulent [illegible] *premature* [illegible] tracted and produces a chain of signs and symptoms which have come to be regarded as pathognomic of bronchiectasis Dilated bronchi surrounded by airless lung may lead to recurrent attacks of pneumonia, but whether this is due to a lighting up of a dormant infection or to a superadded infection cannot be stated with any certainty

A low-grade infection may produce excessive granulations in the dilated bronchi and these granulations may, on occasion, *bleed, giving rise to repeated haemoptyses* Finally bronchiectasis being part of a widespread compensatory mechanism, of which displacement of the heart and emphysema are the other manifestations, dyspnoea may be the outstanding symptom This is especially so in the case of the older patient

The symptoms of the *moderately severe infection leading to chronic ill health* are striking Repeated respiratory crises in childhood are followed by chronic cough and copious sputum, which varies in amount from 1 ounce to 1 pint per day The character of the sputum is muco-purulent as a rule, and *in certain* cases is offensive, a peculiar sweet offensiveness, said to be characteristic of the disease With this productive cough is a series of haemoptyses, as a rule not great in amount but increasing in severity In a case such as that just described, dyspnoea is always a marked feature, although not the primary complaint Further inquiry into the *incidents in* the past may reveal a rib resection for

empyema, a tracheotomy for supposed laryngeal obstruction, or an operation for tuberculous glands in the neck. Examination of the patient usually reveals a thin, wasted, slightly cyanosed person, who is dyspnoeic and whose conversation is punctuated by a productive cough. Clubbing of the fingers and cyanosis of the finger-tips are often present, and on examination of the chest there is wasting with recession of the intercostal spaces, a poor chest movement (as a rule the apex beat is neither visible or palpable) and the trachea is central. Percussion note is hyper-resonant at the apices, and the note at the bases is variable. Patchy dullness may be present, though hyper-resonance is just as likely.

On auscultation the first, and most noticeable, finding is the presence of râles of all calibres over all areas of both lungs, but more marked at the bases. Should the patient get rid of a good deal of sputum during the examination the air entry, which previously may have been poor, becomes loud and bronchial in character.

Further examination of such a patient will show the clubbing of the toes and possibly the presence of some signs of amyloid disease, albuminuria or enlargement of the liver.

X-ray examination of the lungs will show widespread changes throughout the whole of both lungs, with mottled opacities which are more markedly present at the bases. Bronchography as a rule is not very satisfactory, the patient's tolerance to intratracheal interference being low and any Lipiodol introduced is so diluted by the sputum that no clear picture of the degree of the bronchial obstruction is obtained. Post-mortem examination, however, reveals the widespread nature of the complaint.

Cases of this nature usually originate in severe attacks of whooping-cough.

Cases of less marked degree, and especially in which one lower lobe (usually the left) is affected are more common, and some of them exhibit a syndrome closely allied to that already outlined. In such cases, however, the physical signs are more localized and mediastinal displacement is obvious. The heart is displaced to the left. There is a small area of dullness close to the spine posteriorly, with a hyper-resonant note outside this area. Breath sounds are weak in the axilla, and may be absent over the dull area. Coarse rales are present, confined to the area of dullness. If such a patient is then tipped head downwards he will produce, without much effort, 3–4 ounces of thick pus, which may or may not be offensive. The air entry over the dull area will then be loud, and the breathing cavernous in character.

It is rare to find the dilatations confined to one lobe. When the left lower lobe is bronchiectatic, the lingular division of the left upper lobe is usually involved and frequently there is some involvement of the right lower lobe. Occasionally cases are seen in which the middle lobe bears the brunt of the disease and then the corresponding bronchi on the left side may be involved as well. Sometimes cases of collapsed lobes with dilated bronchi are discovered in persons in whom the only symptoms are those of bronchitis. These cases should not be regarded as cases of infected bronchiectasis. The cases of infected bronchiectasis are those in which the infection has spread through the bronchial wall and is located in the atelectatic lung tissue. It is this infection that gives rise to the toxaemia which is characteristic of the disease. This smouldering infection may occasionally burst into flame, giving rise to an illness closely resembling lobar pneumonia

Patients are also encountered who complain of cough and sputum in damp and foggy weather, but who are symptomless *in spells of fine weather This is* such a common occurrence in patients with normal bronchi living in large cities that it alone should not be regarded as being directly connected with the bronchial dilatation

Occasionally patients with a history of productive cough of some years' standing will say that they have had 4 or 5 attacks of pneumonia This history is of great significance, especially if the same lobe of the lung has been involved in each attack of pneumonia

In some of the series of observed cases of bronchiectasis, sputum has dated from a recent acute upper respiratory infection whilst it has been known that the *bronchial dilatation has been present for a great many years but hitherto has* been virtually symptomless

Cases are also seen in which the only complaint is that of recurring haemoptysis As a rule *clinical examination of these patients does not reveal any gross abnormality* though an area of bronchial breathing and a few râles may be discovered *Radiological examination, however, may indicate a localized abnormality sufficient* to warrant bronchography

Other cases are detected by the routine investigation of certain clinical and radiological abnormalitie [illegible] pneumonia, if completely investigatec [illegible] Numbers of cases of asthma, in [illegible] ered, again reveal dilated bronchi Post-operative lung conditions, if persistent, will often show a similar state of affairs

Radiological appearances

The important radiological signs are (a) those of the other compensatory mechanisms secondary to collapse, and (b) the patchy opacities associated with infection in the atelectatic lung

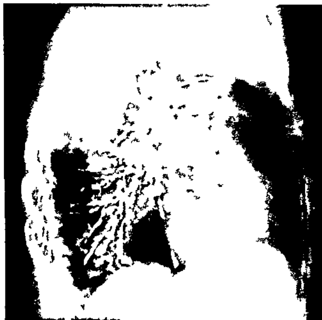
In the early stages, and when the collapse is of recent origin, the atelectatic area may throw a shadow on the x-ray film, and the shape of the shadow is characteristic for the area involved *In cases of longer standing, no shadow of* the atelectatic area will be seen, and the radiological appearances will be those of the compensatory mechanisms, that is, elevation of the corresponding half of *the diaphragm with limitation of movement*, a localized area of emphysema, and displacement of the mediastinum to the affected side (and this displacement of the mediastinum includes such changes as elevation of the pulmonary artery and peaking of the pericardium either on the right or left) Occasionally the condition [illegible] *in the lungs*

If opaq [illegible] se will be found to be crowde [illegible] manifest, and that is the great increase in the size of *the bronchi with inspiration*, and the return to a basic dilated level with expiration

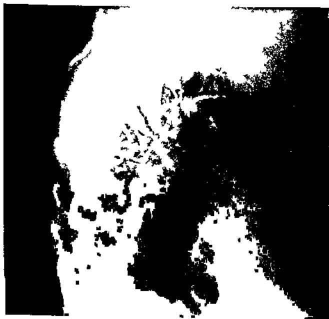
Associat [illegible] there may also *be a move* [illegible] moves to [illegible]

(*see* Figs 59–60)

FIG 59 —Bronchiectasis of long standing Bronchograms showing (*a*) the size of the bronchi in expiration and (*b*) the size of the same bronchi in inspiration



(*a*)



(*b*)



(a)

FIG 60 —(a) Inspiratory and (b) expiratory films in a patient with bronchiectasis of at least 17 years standing showing increase in calibre with inspiration



(b)

It has been customary to classify bronchiectasis according to the bronchographic appearances, and cylindrical, fusiform, and saccular bronchiectasis have all been described These distinctions, although entirely artificial, are of some use clinically, as it will be noted that the cases of saccular dilatation as a rule are the cases of more widespread involvement of the lung, and in which dyspnoea is the outstanding symptom, whereas cylindrical dilatation is as a rule localized

The diagnosis of the condition of bronchiectasis depends on the demonstration of dilated bronchi by means of opaque oil in the bronchial tree The method of introduction of the oil is a matter for the operator's preference Pleas have been advanced for methods where the oil is introduced over the tongue or through the nose, as involving less trauma and less distress to the patient In a large series of cases in which repeated bronchograms were performed by the author and various methods used, the experience was that the patients preferred injection through the crico thyroid membrane, and the only explanation put forward for this is that less manipulation of the tongue and cocainization of the throat is necessary by this method, and that it takes less time

The most important part of any method is the posturing of the patient, so that either the desired area of lung is filled with the radio opaque oil, or preferably, the whole of the bronchial tree is filled on the one side In order to get the most satisfactory and easily interpreted results two separate manipulations are necessary At the first, the diseased part is completely filled, and after an interval, of days or weeks depending on the amount of residual Lipiodol the other side is filled The modern technique is to fill one side completely and then go on immediately to fill the other side, and take oblique x-ray photographs These pictures are somewhat difficult to interpret, as overlapping of the bronchi must occur sometimes, giving the appearance of bronchiectasis where none may be present

The amount of iodized oil used is about 15 20 millilitres This should be sufficient to fill one side of the lung completely, and it is better if used cold (as instructed by the manufacturers) Alveolar filling is annoying, as it obscures the final pictures it is unpredictable and perhaps unavoidable, but the author found it to be less frequent when cold heavy deposit contrast oil was used

Bronchography carried out on children is apt to be disappointing, and alveolar filling is extremely common The explanation of this is probably that the oil is put in under a general anaesthetic, through a bronchoscope, and that the pressure applied through the syringe and catheter causes the oil to be squirted with some force into the finer bronchi

Bronchoscopy is a useful adjunct to bronchography in the diagnosis, and is an essential preliminary to the surgical treatment of bronchiectasis It enables pus to be seen welling out of the bronchi and may help the decision as to which is the most heavily infected of the bronchiectatic areas A fibrous stricture suggests a tuberculous or traumatic origin for the dilatation Unsuspected foreign bodies are sometimes found and can be removed

As part of the preliminary investigations tipping of the patient into the various positions required for drainage of the affected bronchi is essential In the more *grossly infected cases, as a result of tipping, 1 or 2 pieces of thick,* purulent sputum may be obtained The non-production of sputum by this method does not necessarily rule out the presence of infection, and this is especially true in the case of children The children may be held in the appropriate position for minutes at a time and produce no sputum at all, whereas subsequent bronchoscopy or other manipulation may result in a copious production of foul, purulent sputum

Other investigations which are helpful are a white cell count and possibly sedimentation rate In the majority of cases in which infection exists, a leucocytosis is present, and in these cases the erythrocyte sedimentation rate is usually raised *As a rule bronchiectasis is not associated with fever except in the acute* incidents

COURSE OF THE DISEASE

As already indicated, in some cases the infection is so overwhelming that death occurs within a short period of the establishment of the bronchial dilatation These cases more closely resemble an acute lung abscess they were always

acute exacerbation of the lung condition (*see* Figs 61–67) This type of case may develop at any stage a cerebral abscess which in itself may prove fatal

These cases of bilateral involvement have been heavily infected from the beginning They have also been bilateral from the beginning The spread of bronchiectasis from one part of the lung to another *is extremely rare, and when* it does occur, it happens as a result of a plugging of further bronchi, with consequent atelectasis The spread of bronchiectasis by infiltration, such as happens in pulmonary tuberculosis, does not occur

The incidence of infection occurring in a previously collapsed uninfected lung is extremely hard to determine In a series of over 100 cases followed by the author for 13 years, this did not happen in a single instance In the same series, repeated bronchograms were performed, and spread occurred *in one case only,* and th s finally disappeared

vast majority of patients seen and diagnosed in Great Brita
patients followed by the author, 53 per cent married and 31 per cent had children a sequence of events not likely to occur if the patients were as repellent as has been suggested It was found that the pregnancy did not affect the course of the disease, and the disease did not adversely affect the pregnancy



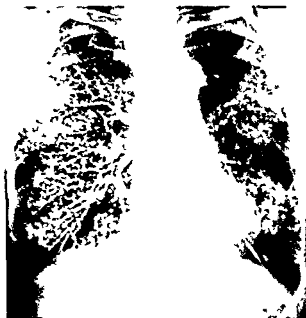
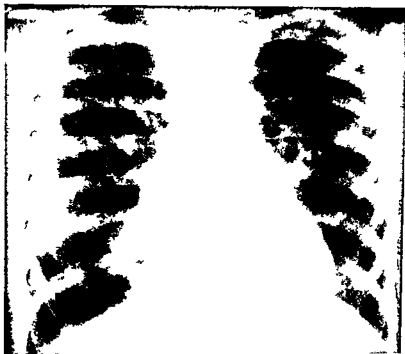


FIG 61 — (*a*) Complete collapse of left lower lobe (1933) with markedly dilated bronchi present in collapsed lobe Both divisions of the lingula are also involved Filling on the right side is poor but there is possibly some dilatation of the bronchus seen close to the vertebral border (*b*) 1948 no appreciable change no further bronchiectasis in left upper lobe and all bronchi outlined on right side are within normal limits except possibly the one seen close to the vertebral border



F G 62 (*a*)



F G 62 (*b*)



FIG 62 —(a) Outline of a collapsed left lower lobe with some abnormally heavy shadowing in the right lower zone (1934), (b) poor filling but confirming the collapse of the left lower lobe and showing dilated bronchi in it (1934) Bronchi elsewhere insufficiently filled, (c) collapsed left lower lobe containing dilated bronchi (1948) The filling of the lingula bronchi is poor but shows what is possibly some bronchiectasis The other bronchi on the left side are normal Bronchi on the right are dilated in the middle lobe but otherwise normal

(c)

Figures in the past have shown an extremely heavy mortality in medically treated cases Most of these studies were in the early years of the use of Lipiodol, and it can only be assumed that the heavily infected cases predominated

Bradshaw, Putney and Clerf (1941) in a series of 171 cases of bronchiectasis followed from 1925 to 1935 had 59 deaths (34 5 per cent) during the period under observation

Perry and King (1940) in a detailed analysis of 144 cases found that 41 per cent died within 5 years of onset and only 15 per cent remained alive for 20 years or longer

Roles and Todd (1933), out of a total of 45 patients treated medically, found that 23 were dead within a short period

COMPLICATIONS

The main complication of bronchiectasis is the recurrence of attacks of infection in the atelectatic lung and in the dilated bronchi The incidence of this complication has never been seriously worked out, but it is undoubtedly high The severity of infection varies from case to case and may be in the nature of a *severe pneumonia, or of a trivial febrile attack associated with an increase in* cough and sputum

Cerebral abscess is listed as one of the most frequent complications, and occurs mainly in the heavily infected patient No case of cerebral abscess in an uninfected patient has been recorded



(a)



(b)

FIG 63 — (a) Complete collapse and bronchiectasis of the left lower lobe (1932) The lingula filling is poor but suggests bronchiectasis The bronchi filled on the right side are within normal limits (b) collapse of the left lower lobe with ectatic bronchi (1948) and bronchiectasis of the lingula The bronchi filled on the right side are within normal limits

Amyloid disease, although it occurs, is not nearly so common as in the corresponding stages of pulmonary tuberculosis

Rheumatism, including infective arthritis, is a rare complication

Nephritis has been reported on several occasions

TREATMENT

The aim of treatment in bronchiectasis must be to rid the bronchi of infection Re establishment of normal calibre is not possible, except in a small number of patients in whom reversion to normal takes place without medical aid The eradication of infection depends on the drainage of the affected bronchi and the administration in one form or another, of drugs designed to kill the organisms prevailing in the sputum

Postural drainage (Fig 68)

The patient must adopt a posture in which the affected bronchi are at a higher level than the bifurcation of the trachea, so that the sputum may run down to this point, whence it can be voided by the patient Thus, when the ventral (or anterior) bronchi are affected, the patient lies on his back, and when the dorsal (or posterior) bronchi are affected, he lies on his face Drainage is also assisted by tilting the patient, so that when the lower bronchi (those below the bifurcation of the trachea) are involved the patient lies with the foot of the bed raised, and when the upper bronchi are involved drainage takes place naturally as the patient is standing As already mentioned, it is unusual to find a case of infected upper lobe bronchiectasis

The drainage is further helped by inclining the patient so that he lies with his uninfected side uppermost Therefore in the case of right dorsal lobe involvement the patient should lie partially on the left side, face uppermost, and in right middle lobe involvement he should lie partially on the left side, face downwards

These postures are easily maintained for a considerable period of time, and consequently are the ones to be recommended in the initial treatment More drastic drainage can be intermittently supplied by making the patient hang over the edge of the bed, with the body inclined vertically downwards—this, of course, for the patient with lower lobe infection This posture cannot be maintained for a long time, and usually produces a severe headache Postural beds and frames have been designed so that the maintenance of these postures can be more easily carried out, and they play a useful part, particularly in cases of [illegible]

hung over the top of the frame

Occasionally, the combination of bronchi involved is such that more than one posture is necessary to produce effective drainage The length of time allowed *for each posture is governed by the extent of the infection in various areas but* it can be stated generally that the lower lobe bronchi, with possibly the middle lobe and lingula bronchi, are more heavily infected than the bronchi of the upper lobe



(a)



(b)

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(*a*)



(*b*)

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FIG 64 — (a) Bronchiectasis of left lower lobe and some ectatic bronchi in left upper lobe including the lingula and probably the pectoral division (1930) The very few bronchi on the right that are filled are within normal limits (b) 1936 similar condition on left side right side is not filled, (c) 1950 no extension of disease The marked spinal curvature is secondary to an attack of poliomyelitis

(a)



(b)



(c)

The second method of procuring drainage is to insert a bronchoscope and apply suction This has been done as frequently as twice a week, as a routine, for years, but is a poor substitute for well applied postural drainage

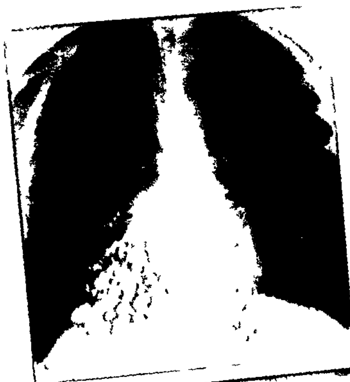
One of the early operations on the lung was designed to drain bronchiectatic cavities through the chest wall This operation has been abandoned for some years but occasionally it has been performed as a result of a mistaken diagnosis of lung abscess Bronchiectatic cavities are multiple it is therefore ineffective

It is sometimes necessary to augment postural drainage by percussion therapy carried out while the patient is in the proper posture, and accomplished by sharply tapping the chest over the affected bronchi for a period of about one minute

Introduction of penicillin and the sulphonamides

In diseases of the lung the two most effective methods of applying penicillin are by intramuscular injection and by inhalation There is still some doubt as to which of these two methods is the more effective and it is really a matter for individual choice Intramuscular injection produces sputum devoid of penicillin sensitive organisms in 4 5 days Inhalation of penicillin produces the same results and is much more impressive to the patient and consequently has a marked psychological effect

Penicillin by injection should be given in doses of 250 000 units 4 times a day and the course should last for at least 2 weeks If ineffective in this time it is



(*a*)

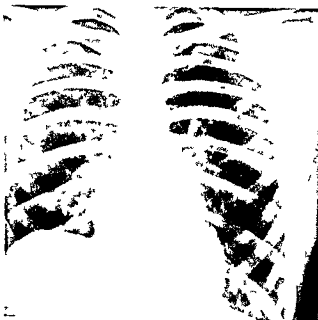
FIG 65 — (*a*) Bronchiectasis of right lower lobe with some dilated bronchi in lower zone of left lung (1927) Filling is too poor to identify these bronchi or to state the condition of lingula (*b*) 1937 similar distribution of bronchiectasis also showing that upper lobes on both right and left side are perfectly normal, (*c*) shows that the bronchiectasis on right side is situated in lower lobe, the middle lobe is normal (*d*) 1945 no gross abnormality apart from a high diaphragm on right side



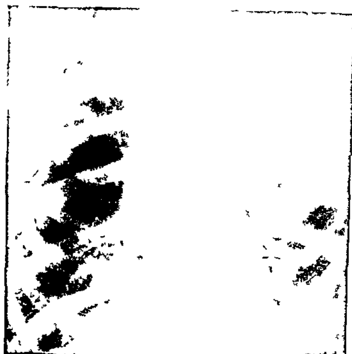
(*b*)



(c)

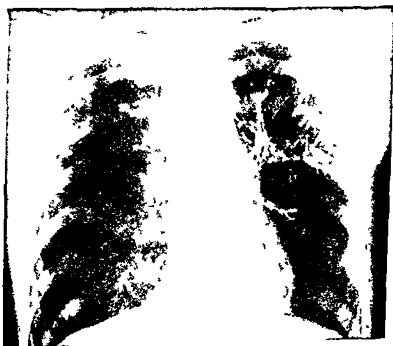


(d)



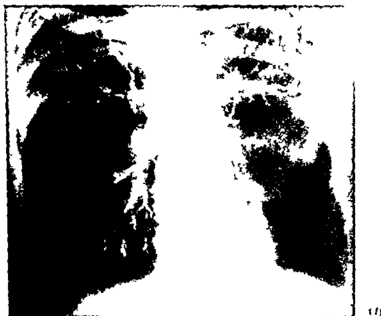
(*a*)

FIG 66—(*a*) Localized emphysema at left base and shadow of collapsed lobe behind heart (1927) (*b*) 1931 confirming this and showing bronchiectasis of left lower lobe and lingula with healthy upper lobe (*c*) 1937 identical condition the bronchi on right side are normal, (*d*) 1948 similar condition though with rather a poor filling



(*b*)

(c)



(f)

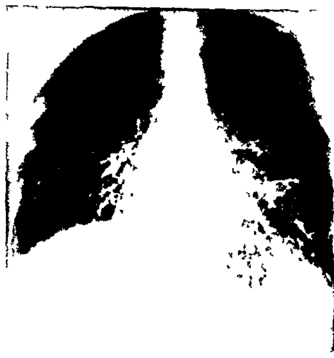


FIG 67 —(*a*) Bronchiectasis of left lower lobe and of the bronchi in the region of right middle lobe (1930) (*b*) 1948 filling is incomplete but all bronchi of upper lobe appear normal (*c*) the right middle lobe appears normal in a right posterior oblique view but there is one ectatic bronchus in the dorsal lobe on right side and also one or two in pectoral division of upper lobe These may be the bronchi outlined in (*a*)

(*a*)



(*b*)


(c)

unl kely to be of any continued benefit but examination of the sputum at the end of the fortnight must be made to find whether the organisms present are insensitive to this form of treatment

If after 2 weeks treatment there has been little decrease in the amount of sputum and the organisms present are found to be Gram negative a short course of streptomycin 0 5 gramme twice a day for 2 days may help to clear the infection

Penicillin by inhalation is given in short spells of about 10 minutes duration during which time 200 000 units of penicill n in 10 millilitres of solution are atomized and inhaled Any form of atomizer is effective though for hospital treatment one of the machines in wh ch the current of air is produced by an electric motor is probably the best These mach nes can be hired for use in the home but there are also some effective hand spray instruments which are inexpensive and can be used and maintained by the pat ent without any great skill or care

Chloromycetin can be used instead of streptomycin 250 milligrams 4-hourly by mouth for 5 days This antibiotic can be used in combination with penicillin therapy in all cases which show a mixed flora in the sputum

Occasionally the sputum is found to contain streptococci or Gram positive organisms which are insensitive to penicillin but sensitive to the sulphonamides For such cases sulphameraz ne 2 grammes 4-hourly for 2 days and then 1 gramme 3 times a day for 2 weeks may be effective

These methods are usually adequate to deodorize the offensive sputum and

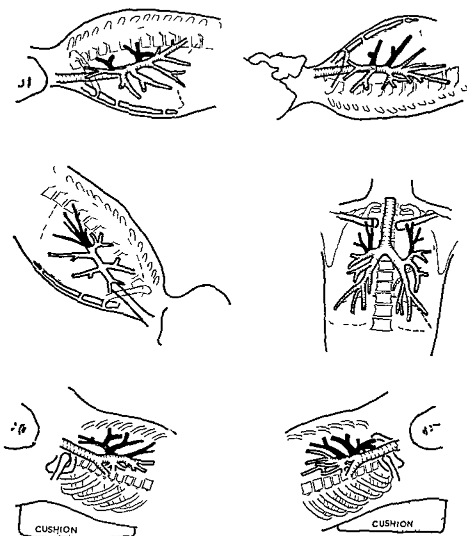


FIG 68 —Positions to be adopted for efficient drainage of the bronchi

also reduce it considerably in bulk However, should the offensive odour persist creosote capsules, 3 grains, 3 times a day by mouth are effective

As a result of these combined forms of treatment, a remarkable improvement in individual cases is recorded The penicillin and sulphonamides are, as stated, usually maintained for 2 weeks, but postural drainage may have to be undertaken for much longer, and indeed in many cases, has to be maintained for life

Indications for surgery

No case should be presented for surgery until a prolonged period of observation and postural drainage has been tried After what length of time bronchiectasis becomes irreversible is not certain and on this score alone 1 year should be allowed to elapse from the date of the first demonstration of the condition before a decision as to surgery is made In that year the case may with postural drainage and antibiotics have become symptomless and even if the dilated bronchi are still present operative treatment is not justifiable

The practicability of the removal of a lung rests with the surgeon, and with the surgeon only The amount of lung which the surgeon is willing to remove

it must be undertaken as a life saving measure The removal of segments of the lung has opened up a wider field, and has rendered operation possible in cases previously judged to be inoperable

In the treatment of bronchiectasis there are several indications for surgery There are certain patients who are so weighed down by the presence of the symptoms even though these symptoms may not be marked that surgery may have to be advised on psychological grounds as this type of patient resents any abnormality which distinguishes him from his fellow men and having to undergo prolonged postural drainage is as repugnant to him as is the 4–5 ounces of sputum

Haemoptysis of gradually increasing severity and of increasing frequency is another reasonable ground for suggesting lobectomy or pneumonectomy

Repeated major infections in the bronchiectatic area constitute another reason for surgery Infection in the bronchi does not except in the case of children, give sufficient trouble to justify lobectomy as the infection can be controlled and probably eradicated by the means already outlined

[illegible]

Certain cases fail to respond to the medical treatment outlined and the infection at the end of the preliminary period is as heavy as it was beforehand Such cases should be re-considered after a period of observation, and if the symptoms persist and there is evidence of septic absorption operation should be advised

Surgery has been advised on several occasions on prophylactic grounds, the prophylaxis being the prevention of spread and of infection in previously un infected dilated bronchi These grounds cannot be justified as it can be shown that infection and spread are neither inevitable nor frequent

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CHAPTER 13

INTRA-THORACIC MANIFESTATIONS OF SARCOIDOSIS

J G SCADDING

DEFINITION

HISTORICALLY the concept of sarcoidosis as a systemic disease has arisen from correlation of partial descriptions each referring only to manifestations in organ, sometimes with passing reference to possible changes in one or more ot organs The syndrome has received a confusing variety of names, includ sarcoidosis, non caseating tuberculosis, pseudo tuberculosis, lymphogranu matosis benigna, and eponyms such as Boeck's disease, Besnier-Boeck's dise and Schaumann s disease The word sarcoidosis has the advantage tha [illegible]
[illegible]
[illegible]
absence of agreement about aetiology, the best criterion of definition is histologi Sarcoidosis may thus be defined as a disease characterized by the presence ir [illegible]

[illegible]

ceeding in the older lesions to conversion into a peculiar hyaline type of fib connective tissue The clinical observations which have led to the concept systemic disorder characterized by this histological picture have been well s marized by Schaumann (1936), Pinner (1938), Snapper and Pompen (1938) Longcope (1941)

The possible manifestations and combinations of manifestations of sarcoid are numerous The skin may be involved in several ways including sarcoids of Boeck and the lupus pernio of Besnier There may be localize generalized enlargement of lymph nodes, and enlargement of the spleen liver usually shows microscopic lesions, but these relatively rarely give ris clinically evident enlargement or to symptoms The eyes may show a chr irido-cyclitis which may be associated with enlargement of the salivary gl giving rise to Heerfordt's syndrome or uveo-parotid fever The small b of the hands and feet may show the changes of Jungling's osteitis tubercul multiplex cystoides Rarer manifestations include endocrine changes, not diabetes insipidus from lesions of the pituitary ; and various forms of involve of the central nervous system, of the kidneys, of the heart and of the gastro-intes tract The lungs and intra thoracic lymph nodes are involved in a [illegible]

[illegible]

the other hand, the lung lesions may give rise to symptoms, notably to dyspnoea, and the course of the illness so produced may be progressive and fatal

INTRA-THORACIC MANIFESTATIONS

The natural history of the intra-thoracic changes in sarcoidosis can best be observed by a study of the radiographic changes The earliest intra thoracic lesion seems in most cases to be hilar lymph-node enlargement, which is usually, though not always, bilateral (Fig 69) This is shortly followed, or accompanied, by a more or less diffuse pulmonary infiltration varying from a fine micro-nodular type resembling miliary tuberculosis (Fig 70) to coarser irregular mottling (Fig 71) Possibly some cases start with the pulmonary infiltration without a preceding stage of hilar lymph-node enlargement Infiltrations of either type may either clear completely so far as radiographic appearances are concerned, or persist for an indefinite time with a variable amount of replacement by fibrosis This fibrosis most frequently takes the form of irregular coarse strands, usually in the middle zones, with emphysematous changes elsewhere (Fig 72) The fibrotic and emphysematous changes give rise to severe and usually progressive dyspnoea, and the clinical picture resembles that of advanced emphysema, giving rise to right ventricular hypertrophy, the cardiovascular syndrome of cor pulmonale, and eventually death from right ventricular failure often precipitated by an intercurrent respiratory infection In the cases which present the picture of fibrosis and emphysema, tubercle bacilli may eventually appear in the sputum, although the radiographic appearances may show little corresponding change Cor pulmonale and the appearance of tubercle bacilli in the sputum may be associated in the terminal stages Occasionally even in cases of sarcoidosis in which it is impossible to demonstrate tubercle bacilli in the sputum and the Mantoux reaction remains negative so that classical caseating tuberculosis seems improbable, cavitation of the lung lesions may appear This may be due to secondary infection giving rise to intercurrent lung abscess, to infection of emphysematous bullae, or to ischaemic breakdown of large tracts of avascular connective tissue In cases of this sort which have been examined *post mortem* the walls of the cavities have usually shown no specific histological change, consisting mainly of fibrous tissue

AETIOLOGY

The aetiology of sarcoidosis remains a subject of controversy, especially on the question whether it should be regarded as tuberculous or non tuberculous There are arguments on both sides The persistent failure to find tubercle bacilli even in patients with extensive lesions, and the frequent lack of sensitivity to tuberculin are the principle points against a tuberculous aetiology On the other hand, there are many observations arising out of a long-term study of patients with pulmonary lesions which strongly suggest a relation to tuberculosis As noted above a considerable proportion of those patients who develop the late fibrotic stage of the disease eventually produce tubercle bacilli in their sputum Cases are recorded in which tubercle bacilli were found in sputa from such patients taken at a time when the Mantoux reaction was still negative, and in some patients who have had persistently negative Mantoux reactions and in whom bacilli have

not been found during life, they have been demonstrated, albeit with difficulty, at necropsy. There is no clear distinguishing characteristic between chronic miliary tuberculosis and diffuse pulmonary infiltrations of sarcoid type; every gradation between the two extreme types occurs. Many reports of post-mortem examinations have been published in which typical sarcoid lesions were present in some organs, typical caseating tuberculous lesions with bacilli present in other organs, and intermediate types of lesions elsewhere. The evidence thus appears [illegible]

[illegible] definition of sarcoidosis, has up to now been proved to be capable of causing lesions of sarcoid type; this obviously does not exclude the possibility that some such agent may be found in future.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Where there are other manifestations of sarcoidosis, particularly skin lesions, generalized lymph adenopathy, and irido-cyclitis with or without salivary gland involvement, the diagnosis of accompanying intra thoracic changes usually presents little difficulty. Difficulty is most frequently met in those cases in which the presenting manifestation is a pulmonary infiltration.

In such cases the conditions which most frequently have to be considered in differential diagnosis are tuberculosis, either of chronic miliary type in the diffuse infiltrations, or of old burnt out fibrotic type in the later stages of pulmonary sarcoidosis; industrial lung disease, notably that due to exposure to beryllium compounds; diffuse nodular fibrosis of the lungs of obscure aetiology; and occasionally fungus diseases of the lung. Histology is the only objective method of supporting a clinical diagnosis of sarcoidosis; but since the definition here adopted demands that the characteristic changes should be present in all affected tissues, the discovery of such changes in single biopsy specimens is not absolute proof of the diagnosis, but is evidence to be added to clinical, radiological and other observations. In all cases a careful search should be made for superficial lymph nodes; in cases of intra thoracic sarcoidosis lymph nodes above the clavicles, especially just above and behind their inner ends, and in the axillae are often found to be palpable on careful search. It is always worthwhile obtaining biopsy of any clinically enlarged lymph node from any site in a suspected case of sarcoidosis. When no palpable lymph nodes can be found, aspiration liver biopsy, even though there is no clinical evidence of involvement of that organ, often shows characteristic lesions.

In differential diagnosis from caseating tuberculosis, histological evidence is not incontrovertible, since it is well recognized that lymph nodes at a distance from a focus of caseating tuberculosis may show non-caseating tuberculous lesions indistinguishable from those of sarcoidosis. The points which suggest a diagnosis of sarcoidosis are a general condition better than would be expected in a patient [illegible]

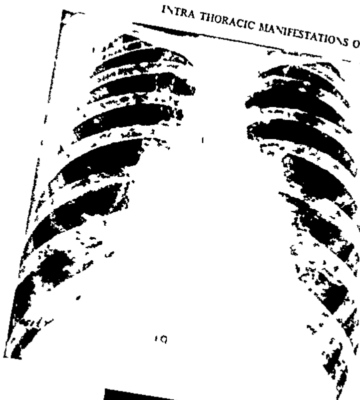


FIG 69 — Enlarged hilar lymph nodes in the early stages of pulmonary sarcoidosis There are also some rather indefinite abnormal shadows in the lungs



FIG 70 — Fine diffuse mottling due to pulmonary sarcoidosis with slight enlargement of hilar lymph nodes

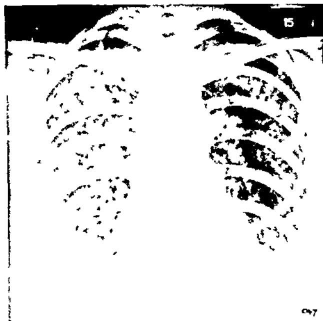


FIG 71 — Coarser diffuse mottling without obvious enlargement of hilar lymph nodes due to pulmonary sarcoidosis

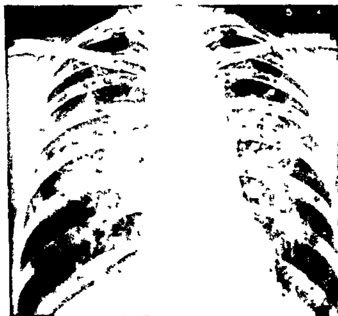


FIG 72 — Late stage pulmonary sarcoidosis with fibrosis in middle zones of lungs and emphysematous changes elsewhere in the lungs. In the original film the residue of diffuse pulmonary mottling was evident (same case as in Fig 69 five years later)

Adopting a purely histological definition of sarcoidosis, it is found that a high proportion of cases have negative Mantoux reactions, often to as much as 10 milligrams of old tuberculin, on the other hand moderate sensitivity, say to 1 : 1,000 old tuberculin, cannot be regarded as incompatible with the diagnosis. It is often the benign course of the disease which determines the label to be applied, so that the application of the term sarcoidosis might be regarded as indicating a prognosis rather than a diagnosis.

In all cases showing diffuse pulmonary infiltrations a careful and complete occupational history must be taken. Difficult diagnostic problems arise in cases with a possible exposure to noxious dust and radiographic changes in the lungs which could be interpreted as due to sarcoidosis. In such instances all available evidence must be examined, and it is sometimes only after a period of observation that an opinion can be given. Special difficulty arises in the case of beryllium granulomatosis of the lungs, since in this condition the histological changes as well as the radiographic appearances and symptoms are similar to those of sarcoidosis, in fact on a histological definition of sarcoidosis it would be logical to refer to these cases as beryllium sarcoidosis. Estimation of the beryllium content of the urine (which is a specialized procedure) may be helpful in such cases.

Mantoux-negative erythema nodosum

An interesting group of cases of erythema nodosum with bilateral hilar lymph-node enlargement and negative Mantoux reaction appears to have a relation to sarcoidosis. This group is clearly demarcated from the more usual type of erythema nodosum associated with primary tuberculous infection, in which there may be hilar lymph-node enlargement, but this is characteristically unilateral, and the Mantoux reaction is invariably strongly positive. In cases of Mantoux-negative erythema nodosum with bilateral hilar lymph-node enlargement, diffuse pulmonary infiltrations, which generally prove to be transient, are frequently seen, and in a few cases biopsy of enlarged superficial lymph nodes has shown histological changes of sarcoidosis.

PROGNOSIS OF THE LUNG LESIONS

In the early stage of diffuse pulmonary infiltration it is impossible to give an accurate forecast of the probable course in the individual case. It certainly depends to some extent upon the patient's race, in Negroes a progressive course possibly culminating in frank tuberculosis is more frequent than in Europeans. A survey of published figures suggests that where intra-thoracic manifestations are a leading feature, regression of the lesions amounting to complete clearing may occur in about half, sometimes after the lesions have been apparently stationary for a considerable time. Of the remaining half some will show very little change after relatively prolonged periods of observation, but some will show increase in the extent and severity of the lesion with eventual development of fibrosis and secondary emphysema, and some of this last group will eventually produce tubercle bacilli in the sputum. Death in such cases may occur after prolonged illness from right heart failure or from intercurrent respiratory infection. It is obvious that if a patient first comes under observation with the late fibrotic and emphysematous changes, no permanent improvement can be expected.

TREATMENT

No form of treatment can be relied upon to influence the course of the disease In the more acute types approximating to chronic miliary tuberculosis it may appear reasonable to try the effect of streptomycin However, streptomycin is most effective in the acute exudative types of tuberculosis and relatively little, if at all, effective in the most productive types, and the lesions of sarcoidosis are the perfect example of the productive non exudative reaction The relatively little information at present available about the effect of streptomycin in sarcoidosis is in accordance with expectation based on these considerations Calciferol has been used in an attempt to modify the course of some of the progressive types of nodular infiltrations Since in these forms of pulmonary sarcoidosis spontaneous regression is frequent results are difficult to interpret, and evidence is as yet inconclusive about the value of this form of treatment Many patients with sarcoidosis are peculiarly susceptible to the toxic effects of calciferol and may suffer serious biochemical and symptomatic disturbances with evidence of renal damage after the uncontrolled administration of quite small doses At present, therefore calciferol should not be given to patients who are symptom free and whose lesions do not seem to be progressive, its use may possibly be justified in those patients who have diffuse pulmonary infiltrations which are increasing in density under observation In such cases, after preliminary investigation of the levels of serum calcium and blood urea and of renal function, 50 000 units daily by mouth should be given at first This may be increased progressively if there is neither symptomatic nor serious biochemical disturbance up to a maximum of 150 000 units daily and the tolerated dosage maintained for 4 months Minor upsets during the course of treatment may necessitate adjustment of dosage, and some patients cannot tolerate even 50 000 units daily Radiographic improvement usually is not evident until after the end of this course of treatment

For the later stages of pulmonary sarcoidosis with permanent fibrotic and emphysematous changes no curative treatment is possible, and the treatment is similar to that of a case of pulmonary emphysema and fibrosis of any other cause

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Resultant or associated pulmonary disease—Broncho-pneumonia is usual in the terminal stages of all reticuloses Pulmonary tuberculosis is not infrequently seen in association with chronic leukaemia or lymphadenoma and difficulty may be experienced in deciding which disease came first Pulmonary cavitation occurring during the course of a reticulosis may be due to tuberculosis, to a lung abscess or, occasionally, to the breakdown of a mass of lymphadenomatous glands

LYMPHADENOMA AND LYMPHOSARCOMA

In lymphadenoma, abnormal rad[illegible]
rather more than 50 per cent of [illegible]
glands or pleural effusion In r[illegible]
filtrated in one or other of the ways described above (Peirce, Jacox and Hildreth, 1936, Gall and Mallory, 1942) The rate of progression is slow and the results of radiotherapy, other than for enlarged mediastinal glands, is disappointing A sudden deterioration of health, with rapid increase in the lesions, suggests sarcomatous degeneration (*see* Figs 75–76)

In lymphosarcoma the radiographic changes are similar to lymphadenoma but occur in only about one case in five They progress rapidly and usually show a favourable initial response to radiotherapy (Fig 77)

MYCOSIS FUNGOIDES

The disease may be limited to the skin for many years and then rapidly extend to other parts of the body, the lungs may show large roundish masses or a coarse mottling, as a result of haematogenous dissemination (*see* Fig 78)

THE LEUKAEMIAS

Acute leukaemia

In the acute leukaemias, which run a febrile course and terminate fatally within a few months, pulmonary infarcts and venous thromboses are common, an aspiration pneumonia, perhaps with an acute lung abscess, is likely to occur particularly in those patients, comprising about one-third of the total number, who have an associated acute ulcerative infection of the mouth or throat The respiratory symptoms of these complications are often surprisingly slight and may be overlooked In children with acute lymphatic leukaemia massive mediastinal glandular enlargement may develop (*see* Figs 79–80)

Chronic leukaemia

In the chronic leukaemias about one case in five shows an abnormal radiograph of the chest, in chronic lymphatic leukaemia the changes are indistinguishable from those of lymphadenoma, enlarged mediastinal glands being the commonest feature (*see* Fig 81), in chronic myeloid leukaemia the radiological signs are usually slight and inconclusive (*see* Kirklin and Hefke, 1931, Falconer and Leonard, 1938)

OTHER DISEASES OF THE RETICULO ENDOTHELIAL SYSTEM

Gaucher's disease—This rarely extends to the lungs Merklen, Waitz and Warter (1933) were able to recover Gaucher's cells from the sputum of an advanced



FIG. 75 — Lymphadenoma with enlarged mediastinal glands and rounded deposits in the lungs



FIG. 76 — Lymphadenoma with extensive involvement of the lungs and no radiological evidence of enlarged mediastinal glands. The invasion had been progressive during the previous year and failed to respond to radiotherapy



FIG 77 — Lymphosarcoma with massive glandular enlargement Following radiotherapy the mass disappeared radiologically to recur 3 years later



FIG 78 — Mycosis fungoides section of the whole of the right lung shows many deposits An extensive eruption of the skin had been present for 6 years and a radiograph of the chest had been normal 3 months before death



FIG 79 — Acute lymphatic leukaemia in a boy aged 13 years showing massive mediastinal glandular enlargement He died after an illness of two months



FIG 80 — A lateral view of the case shown in Fig 79 The characteristic enlargement of the glands into the anterior mediastinum is seen

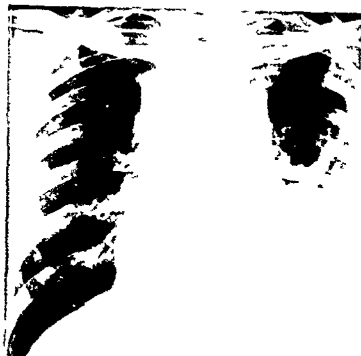


FIG 81 — *Chronic lymphatic leukaemia with enlarged mediastinal glands and a left pleural effusion*

case, and Myers (1937) found considerable involvement of the lungs and mediastinal glands in another

Niemann Pick s disease —In this disease the lungs may be involved in the general process of congestion and oedema and the alveoli may contain many of the characteristic large pale cells (Canmann, 1944)

Other syndromes —Hand Schuller Christian disease Letterer Siwe s disease and eosinophilic glanuloma of bone are described separately under the heading of Honeycomb Lungs

LEUKAEMOID REACTIONS IN PULMONARY DISEASE

Leukaemoid reactions are occasionally seen in diseases of the lungs They consist in a leukocytosis with immature cells and need to be differentiated from leukaemia Forkner (1938) states that in leukaemia there is almost invariably a thrombocytopenia and that the immature forms are of a uniform type and comprise at least 70 per cent of the white blood cells neither of these features is seen in a *leukaemoid* reaction In whooping cough white cell counts of 100 000 per cubic millimetre and more have been recorded usually in cases complicated by broncho pneumonia *nearly all the cells being lymphocytes Pneumonia in children may provoke* counts of 50 000–100,000 white cells per cubic millimetre often including many immature forms , the counts rapidly return to normal during convalescence

The reaction in miliary tuberculosis —A leukaemoid reaction is fairly common in miliary tuberculosis and should be suspected when there is enlargement of the liver,

spleen or lymph glands, the abnormal blood cells are nearly always of the myelogenous series At autopsy the affected organs show miliary tubercles but none of the changes typical of a true leukaemia The reaction is rare in the more chronic forms of tuberculosis

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CHAPTER 15

HONEYCOMB LUNGS

NEVILLE OSWALD

AETIOLOGY

AMONGST the many varieties of pulmonary cysts, instances are occasionally seen of lungs which on cross section resemble a honeycomb or sponge, as a result of cysts uniformly distributed throughout both lungs and measuring up to about 1 centimetre in diameter The cysts may be due to a primary pulmonary disease or be part of a general medical disorder , in either case the clinical features are similar , spontaneous pneumothoraces being common and right heart failure almost invariably the cause of death The condition has been reviewed in an article by Oswald and Parkinson (1949)

Pathogenesis —There is no doubt that in a minority of instances a developmental defect is the causative factor It is equally certain that diffusely infiltrating

occasionally be the cause

DEVELOPMENTAL GROUP

The developmental group is in many ways the most typical and the most easily walled, tend to be rather large and give
ls in otherwise translucent lung fields
in childhood or early adult life ; they may be recurrent or bilateral and are sometimes fatal Other causes of death include right heart failure, broncho-pneumonia and associated developmental defects Few patients live for more than 30 years Congenital heart disease is a not infrequent concomitant (*see* Figs 82 83)

The mode of production of the cysts is *unknown* Pulmonary cysts of any kind are excessively rare in still-born foetuses and for this reason, apart from any others, the term " congenital cystic disease " should be abandoned Doubtless there is a developmental factor, which may not necessarily operate until some *months or* years after birth The problem as it presents is to account, often, for a *child with* well developed cysts and no previous history of respiratory disease, in the light of the knowledge that such cysts have hardly ever been observed at birth In the present state of our knowledge it is impossible to state whether the cysts are truly congenital or whether they have been caused by a trivial respiratory infection after *birth in* lungs of poor quality The characteristically thin walls of the cysts, the paucity of reaction in the connective tissues of
developmental abnormalities elsewh
entity and to justify their being label

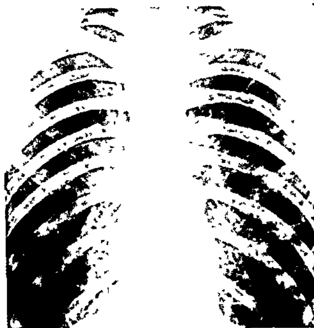


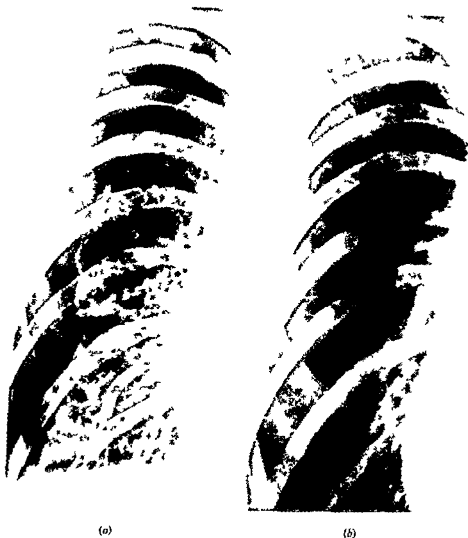
FIG 82 — Developmental type showing fine linear striations through both lungs A man aged 23 years who had several spontaneous pneumothoraces from the last of which he suddenly died

TUBEROUS SCLEROSIS

Classification

Tuberous sclerosis, in its classical form, is seen as a familial disease of children, mental deficiency, epilepsy and adenoma sebaceum of the face being its commonest manifestations There may in addition be myomas of the heart and kidneys, sub-ungual fibromas and tumours of the retinae Such children rarely survive until adult life and none have so far shown any pulmonary lesions In recent years some half-a-dozen adults, all females and some with a family history of tuberous sclerosis, have been found to have a diffuse leiomyomatous infiltration of both lungs with honeycomb cysts (*see* Fig 84), none has had mental deficiency or epilepsy, but all except one have had adenoma sebaceum and sub-ungual fibromas and two showed the typical involvement of the cerebral cortex and kidneys at autopsy

There is thus a small group of patients with somewhat atypical tuberous sclerosis whose symptoms are predominantly respiratory Closely related to these are others with identical pulmonary changes and with evidence of extra-pulmonary mesodermal dysplasia, but with none of the characteristic clinical signs of tuberous sclerosis They would appear to belong to a range of diseases having tuberous sclerosis at one end and the angiomatoses of Sturge-Weber and Hippel-Lindau at the other, they may conveniently be regarded as cases of multifocal mesodermal dysplasia A third group comprises cases with a vast increase of muscular tissue in the lungs and with honeycomb formation, but with no extra-pulmonary lesions



(a) (b)

Fi ° / D

In the past these have been called muscular cirrhosis of the lungs but it is probable that they belong to this group of mesodermal defects and represent a purely pulmonary form

The pathological process in the whole group is similar and consists of smooth muscle growing out from the smaller bronchi and blood vessels often forming irregular masses in the lungs or within the lumina of the bronchi and sometimes directly infiltrating the alveolar walls The mechanism of production of the cysts

is probably by obstruction or strangulation of the smaller radicles of the bronchial tree

Clinical picture and diagnosis

The symptoms derive from spontaneous pneumothoraces and right heart failure, which rarely occur before adult life The radiological appearances are of two kinds Usually a diffuse reticulation appears throughout both lungs, which increases in density over a period of years , the cysts may be difficult to see in the early stages, but they become more apparent as the general density of the lungs increases In the other variety an infiltrating process starts in one or both lungs and gradually spreads, making the differential diagnosis from pulmonary tuberculosis extremely difficult, unless there are extra pulmonary manifestations of the disease or a piece of lung is removed for microscopical examination (Fig 85)

XANTHOMATOSES

Classification

Amongst the xanthomatoses there are three varieties which are closely related and often present in a mixed form , the lungs may be involved in any or in any combination of them and show a diffuse granuloma with honeycombing The three varieties comprise (1) Letterer Siwe's disease, (2) Hand Schuller Christian disease, and (3) eosinophilic granuloma of bone

Letterer Siwe's disease is typically an acute aleukaemic reticulosis of infants, having a non lipoid-containing mononuclear as its characteristic cell, and running an acute febrile course of a few weeks or months, with enlarged liver, spleen and lymph glands, and terminating fatally

Hand Schüller Christian disease tends to develop in early childhood It frequently shows its triad of exophthalmos, diabetes insipidus and erosions of the skull, as a result of invasion by cholesterol-containing mononuclear cells (Fig 86)

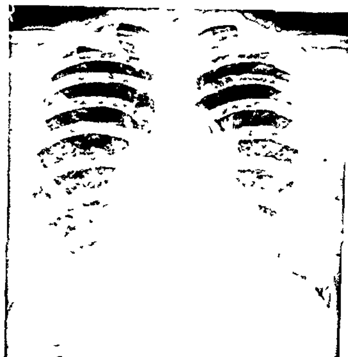
Eosinophilic granuloma may occur in childhood or adult life, and is diagnosed by the finding of cystic spaces, usually in the shaft of a long bone, filled with eosinophil cells

Microscopical examination of sections taken in any of these diseases may show that the lungs have become diffusely infiltrated with a granuloma The characteristic cells may, however, be sparse or difficult to see because of secondary infection There may or may not be honeycombing of the lungs, and it seems extremely probable that the cyst formation represents a late phase

Clinical features

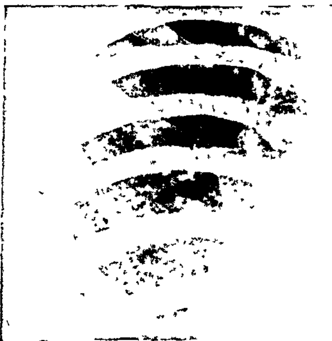
The respiratory symptoms depend largely upon the age of the patient and the clinical course of the extra pulmonary features In infants and young children, bronchitis and broncho-pneumonia are common and although the immediate cause of death may be right heart failure, prolonged fever and inanition are major contributory factors

In a certain number of cases, the xanthomatosis appears to become arrested, and many years later evidence of pulmonary or pituitary insufficiency may be apparent (Oechsli and Miles, 1934) One such case, a man in the late twenties, has been under observation for 5 years with well marked honeycomb lungs which, apart



(a)

FIG 84 —(a) Tuberous sclerosis with diffuse mottling of lungs and a honeycomb appearance becoming visible in the mid zones in a *woman aged 31 years* She also has adenoma sebaceum of the face subungual fibromas of the toes and a filling defect in the left kidney which probably represents a myoma During 4 years' observation she has continued to *lead a busy life as a* shop assistant troubled only by slight breathlessness (b) Higher magnification showing myomatous infiltration of the lung



(b)





(a)

FIG 85 — (a) Irregular type of infiltration which may be seen in tuberous sclerosis in a woman aged 36 years. A spontaneous pneumothorax had occurred 5 years previously and the infiltration starting in left upper zone was seen to spread radiologically during 3 years. She also had adenoma sebaceum of the face. The diagnosis of leiomyomatous infiltration with honeycomb formation was confirmed by lung biopsy. (b) Higher magnification.



(b)

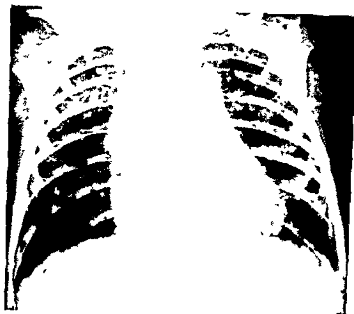


FIG 86 —(a) Hand Schuller Christian s disease in a baby aged 9 months The skull and spine were also affected The wide upper mediastinal shadow represents an enlarged thymus gland confirmed at autopsy There was considerable secondary infection of the lungs at the time of the radiograph (b) section of lung showing the cystic spaces

(a)



(b)

insipidus for 8 years and his honeycomb lungs have been radiologically unchanged for 4 years , in recent months progressive pituitary failure has developed The combination of honeycomb lungs and diabetes insipidus (*see* Fig 89), of which there are some half-dozen examples in the literature, is almost certain evidence of a former xanthomatosis

The radiological changes in infancy consist in a mixture of rather ill defined cysts, reticulation, and signs of pulmonary congestion and broncho pneumonia , the

cysts when present are usually sufficiently obvious to enable a diagnosis to be made. In adults the density of the reticulation varies within very wide limits; in some cases the connective-tissue reaction is so slight that the appearances are very similar to those seen in the developmental type of honeycomb lungs, in others the reticulation is gross and coarse. The cysts can usually be seen without difficulty especially during the phases of spontaneous pneumothorax.

BILIARY CIRRHOSIS OF INFANTS

Biliary cirrhosis of infants, a condition of which little is known, consists in a granulomatous infiltration of the biliary tracts, which gives rise to jaundice and is usually fatal; the pulmonary changes are indistinguishable from those of xanthomatosis.

HONEYCOMB LUNGS OF UNCERTAIN AETIOLOGY

In addition to the above examples of honeycomb lungs occurring as part of various general medical disorders, there are others, numerically representing about 50 per cent of the total, in which the disease is limited to the lungs and the aetiology of which is by no means certain. The group comprises adults, mostly males, ranging in age from 20 to 60 years, with spontaneous pneumothoraces and right heart failure as the outstanding clinical features of the disease and having diffuse radiological changes similar (*see* Fig 90) to those described above as occurring in adults. It is likely that there are several different aetiological factors within the group, but, whatever their nature, they must affect both lungs equally. Pathologically, the features are somewhat similar in all, with varying degrees of inflammation and fibrosis of the connective tissue, and universal cyst formation. The following possible aetiological factors should be considered.

Diffuse cystic granuloma of the lungs

[illegible]

the cysts arise by a process of strangulation of the smaller branches of the bronchial tree, and that their walls become infiltrated by the same process. They are uncerta[illegible]

[illegible]

xanthomatosis lies in the paucity of the characteristic cells, largely as a result of secondary infection. As things stand, in those cases in which there is a diffuse [illegible]

[illegible]

Emphysema

Whether or not universal emphysema should be regarded as a variety of honeycomb lungs is a matter of opinion. Emphysema normally derives from sustained

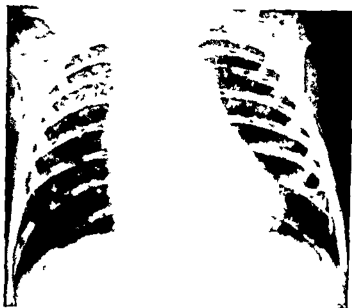


FIG 86 —(*a*) Hand Schuller Christian's disease in a baby aged 9 months The skull and spine were also affected The wide upper mediastinal shadow represents an enlarged thymus gland confirmed at autopsy There was considerable secondary infection of the lungs at the time of the radiograph (*b*) section of lung showing the cystic spaces

(*a*)



(*b*)

from passing through a phase of spontaneous pneumothoraces, have remained unchanged radiologically, in his case severe diabetes insipidus has recently developed (Fig 87) Another man (*see* Fig 88) in his fifties, had a pathological fracture of his femur as a result of an eosinophilic granuloma 10 years ago, he has had diabetes insipidus for 8 years and his honeycomb lungs have been radiologically unchanged for 4 years, in recent months progressive pituitary failure has developed The combination of honeycomb lungs and diabetes insipidus (*see* Fig 89) of which there are some half dozen examples in the literature, is almost certain evidence of a former xanthomatosis

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Diffuse cystic granuloma of the lungs

In a recent pathological study, Cunningham and Parkinson (1950) have observed gradations in the amount of granulation tissue and fibrosis, which would account well for the range of densities seen in radiographs of the chest They consider that the cysts arise by a process of strangulation of the smaller branches of the bronchial tree, and that their walls become infiltrated by the same process They are uncertain of the aetiology of the condition, to which they apply the rather non-committal term, diffuse cystic granuloma of the lungs

There is no doubt that the changes are very similar to, and may be indistinguishable from, those seen in xanthomatosis—a fact which raises the question of the difference between this condition and the "eosinophilic xanthomatous granuloma" of Thannhauser (1947) The difficulty in the diagnosis of solitary pulmonary xanthomatosis lies in the paucity of the characteristic cells, largely as a result of secondary infection As things stand, in those cases in which there is a diffuse granuloma of the lungs without evidence of a xanthoma elsewhere in the body a non-committal term such as diffuse cystic granuloma should be used, in order to permit of an alternative aetiology to xanthomatosis, if necessary

Emphysema

Whether or not universal emphysema should be regarded as a variety of honeycomb lungs is a matter of opinion Emphysema normally derives from sustained

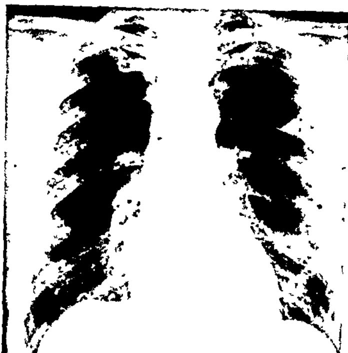


FIG 88 — (*a*) Coarse mottling of the lungs with a honeycomb appearance towards the bases, in a man aged 57 years who also has an eosinophilic granuloma of his femur and diabetes insipidus Radiotherapy to the lungs caused an increase in breathlessness Radiographs of his chest have remained unchanged over 6 years, but he now suffers from progressive pituitary failure , (*b*) Higher magnification

(*a*)



(*b*)

FIG 89 —(a) Diffuse cystic granuloma associated with diabetes insipidus and bilateral spontaneous pneumothoraces in a man aged 38 years The heavy coarseness would appear to represent a late and chronic phase of the disease He died from heart failure a year later Autopsy was not performed (b) higher magnification

(a)



(b)

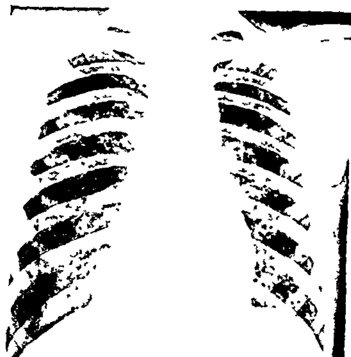
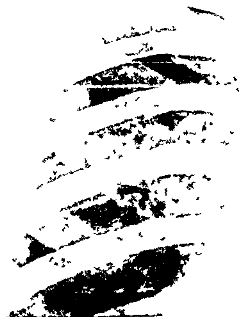
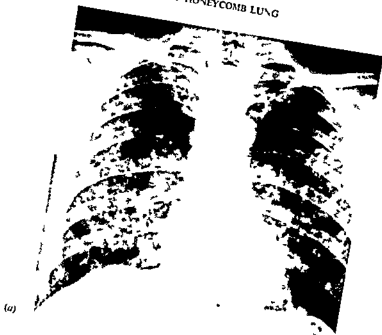
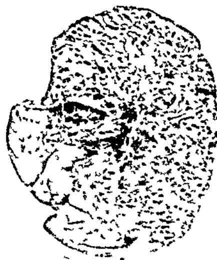


FIG 90 — (*a*) Diffuse cystic granuloma with right spontaneous pneumothorax in a man aged 20 years He died from right heart failure 10 years later autopsy revealing gross granulomatous invasion of the lungs with many cysts (*b*) higher magnification





(a)



(b)

FIG 91 — (a) A chronic bronchitic patient aged 55 years with heart fa lure (b) section of whole lung showing the universal distribution of the cysts the other lung was sim lar

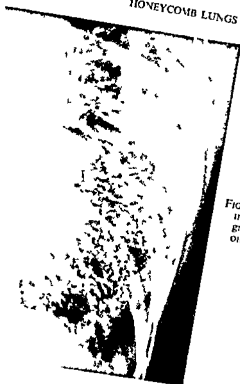


FIG 92—Diffuse cystic granuloma in a pituitary dwarf the bronchogram showing that the iodized oil does not enter the cysts

Spontaneous pneumothorax develops in about 50 per cent of all patients with honey comb lungs

Right heart failure is the usual cause of death in this almost uniformly fatal disease but it may be delayed until late middle age, it is due to a progressive diminution in effective alveolar surface the end frequently being hastened by a secondary respiratory infection

A thorough search should always be made for extra pulmonary features which might lead to the diagnosis of a general disorder having honeycomb lungs as one of its manifestations

Radiological appearances

These differ somewhat in each group and therefore are described separately above In general the lung fields are uniformly marked by cysts and reticulation the latter showing wide variations in density Tomograms are often helpful in demonstrating the cysts On bronchography iodized oil never enters the cysts and occasionally the smaller bronchi appear to be encircling them (*see* Fig 92) The diagnosis is often easy from a single radiograph especially during a spontaneous pneumothorax when the cysts stand out clearly In the presence of heart failure or respiratory infection the widespread distribution of the cysts may not be demonstrable there is thus a group of patients in whom honeycomb lungs may be suspected radiologically but to whom the clinical label cannot be applied without pathological proof

Differential diagnosis

The differential diagnosis of diffuse pulmonary reticulation is formidable, it includes pneumoconiosis, sarcoidosis, chronic miliary tuberculosis, lymphangitis carcinomatosa, and other rare diseases, in none of which have honeycomb lungs ever been found

TREATMENT

Aside from the treatment of such general disorders as may be present, the successful management of spontaneous pneumothoraces is often a life-saving measure [illegible]

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CHAPTER 16

CARDIO-PULMONARY SCHISTOSOMIASIS

D Evan Bedford

Synonym Bilharzial heart disease

Pulmonary schistosomiasis with its cardiac complications may be due to either *Schistosoma haematobium* or *S mansoni*, and is always secondary to urinary or intestinal infestation Pulmonary lesions were found in 33 per cent of 282 cases of schistosomiasis examined at necropsy at the Kasr-el-Aini Hospital, Cairo (Shaw and Ghareeb, 1938), and they have also been reported from other countries where bilharzial infestation is prevalent, namely North and South Africa, South America and the West Indies Our knowledge of cardio pulmonary schistosomiasis is based mainly on the disease as encountered in Egypt, where Bilharz first discovered the parasite, where its pathology has been specially investigated by Sorour (1932) and by Shaw and Ghareeb (1938), and where its clinical manifestations were first recognized by Azmy (1932), Mainzer (1935) and

in Porto Rico

PATHOLOGY

Pulmonary lesions may be caused either by the worms themselves or by the ova, the latter being by far the more important cause

Invasion by ova

Ova derived from the systemic or mesenteric veins pass through the right heart to reach the lungs as emboli, lodging in the arterioles which accompany the respiratory bronchioles *In the case of S haematobium, the ova are derived* from the vesical, ureteric, prostatic and uterine venous plexuses and pass *via* the internal iliac veins to the inferior vena cava , in the case of *S mansoni*, the ova are derived from the portal venous system and reach the vena cava *via* dilated porto caval anastomoses due to cirrhosis of the liver (Egyptian hepato-splenomegaly)

The ovum, once impacted in a pulmonary arteriole, produces a necrotizing arteriolitis in which the continuity of the media is destroyed, allowing the ovum to penetrate the vessel wall, outside which is formed a " bilharzial tubercle ", not unlike the tubercle of Koch's infection In the early stages there is a cellular reaction around the ovum, consisting of eosinophil leucocytes, endothelioid cells, giant cells, and later lymphocytes *This cellular reaction subsides*, leaving a nodular scar containing the calcified or disintegrated ovum The damaged arteriole is occluded by intimal thickening without thrombosis, and the occluding tissue becomes recanalized Owing to focal destruction of the media, the newly



FIG 93—Sect on showing th ckened pulmonary arter ole w th adjacent nodule conta n ng a g ant cell fill ng the shell of a bilharzia ovum (*From Bedford A daray and G g s* 1946 *by co rtesy of B t Heart J*)

In the course of time repetitive embolization leads to widespread arterial obliteration sufficient to cause pulmonary hypertension with consequent hyper trophy of the right ventricle and gross dilatation of the pulmonary arterial tree Both the main pulmonary stem and its hilar branches may reach aneurysmal size and their walls become thickened atheromatous calcified or even thrombosed Bilharzial tubercles have been found in the ventricular myocardium in a case of *S japon cum* infection (Strong 1944) and Kenawy has reported focal cellular infiltration of the myocardium but ova were not identified

Thus the pulmonary lesion is essentially arterial and the lung parenchyma is little affected apart from the perivascular tubercles A broncho pulmonary form has been recognized radiolog cally and evidently occurs in the earlier and more active phase of the disease when cellular reactions around the ova are present Sorour (1932) has described an endobronchitis obliterans of the larger and medium sized bronchi but gross fibrosis of the lungs does not occur

Invasion by worms

Sometimes the worms themselves—either the male or coupled male and female—reach the lungs as emboli where they cause arterial thrombosis and a focal verminous pneumonia the foci of consolidation being 1 5 millimetres in diameter Day (1937) has reported finding pulmonary arteries packed with coupled worms



FIG 94 — Drawing of section showing occluded pulmonary arteriole with revascularization of lumen surrounded by angiomatoid formation characteristic of bilharzial pulmonary endarteritis From same case as Fig 96 (*From Bedford, Aidaros and Girgis*, 1946 *by courtesy of Brit Heart J*")

When the inflammatory reaction subsides, the dead worm becomes calcified and enclosed in a fibrous capsule

CLINICAL FEATURES

Incidence

In Egypt, cardio-pulmonary bilharziasis is encountered almost entirely amongst the agricultural workers inhabiting the Nile Delta, the majority of whom are infested with bilharzia The pulmonary lesion is commonest in males aged 12-35 years, and is always associated with either urinary or intestinal bilharziasis, in the latter case hepato-splenomegaly is usually present

Pulmonary disease

Before the heart is seriously involved, asthmatic or bronchitic symptoms may lead to radiography of the chest, which shows a miliary mottling of the lung fields, not unlike that of miliary tuberculosis or pulmonary haemosiderosis, as described by Mainzer (1935 and 1938) and by Erfan and his colleagues (1949) The pulmonary mottling is due to dilated arterioles beaded by nodules about 1



Fig 95 — Radiograph from a case of pulmonary schistosomiasis in active stage showing mottled lung fields due to beaded and dilated vessels There is some dilatation of the main pulmonary artery and its hilar branches (From Erfan and his colleagues 1949 by courtesy of Trans R Soc trop Med Hyg)

millimetre in diameter (Fig 95) these are due presumably, to bilharzial tubercles The lung changes may be evident before the heart is much involved or may be associated with enlargement of the heart and main pulmonary vessels Emphysema, with basal bronchitic or catarrhal signs, is found in some cases, but more often clinical signs in the lungs are absent An acute focal reaction in the lungs with increased clinical or radiological signs may result from antimony treatment

Bilharzial heart disease

In the cardiac phase of the disease the clinical picture is that of pulmonary hypertension without gross lung disease The apex is displaced to the left and systolic pulsation is palpable over the pulmonary artery and conus A pulmonary systolic murmur and accentuated second sound are audible and sometimes there is a diastolic Graham Steell murmur due to relative pulmonary incompetence as in Azmy s original case (Azmy 1932) The radiograph shows enlargement of the right ventricle towards the left, and dilatation of the pulmonary trunk and its hilar branches which may pulsate excessively Aneurysmal dilatation of the pulmonary artery is not uncommon (Fig 96) In the writer s case the aorta was hypoplastic as was also noticed by Erfan

Cyanosis is usually absent until heart failure appears and as is usual in primary pulmonary hypertension the arterial oxygen saturation is normal or slightly reduced for example 82–95 per cent in the cases described by Soliman (1950).

Fi [illegible] aged [illegible] its [illegible] the [illegible] *by*

Clubbing of the fingers is inconstant The electrocardiogram shows the pattern of right ventricular hypertrophy (Kenawy 1950) Eosinophilia is sometimes found in the earlier and active stages of the disease but not usually in the cardiac phase In Egypt, bilharziasis is often associated with ankylostomiasis in which case anaemia and eosinophilia are common Eventually congestive failure with regular rhythm occurs and proves refractory to treatment

DIAGNOSIS

Broncho-pulmonary disease

In the broncho pulmonary form radiographs show a diffuse miliary appearance of the lung fields which, in association with evidence of urinary or intestinal bilharziasis or with hepato splenomegaly is suggestive (*see* Fig 95) Ova may be found in the urine or stools, and very rarely in the sputum

Cardiac disease

The cardiac condition with aneurysmal dilatation of the pulmonary artery has

to be distinguished (1) from other forms of pulmonary heart disease, (2) from mitral stenosis with haemosiderosis of the lungs and (3) from congenital heart disease, especially auricular septal defect which may give an identical radiological picture. Mitral stenosis is excluded by the absence of left auricular enlargement as shown by radioscopy in the right oblique position after a barium swallow, and by the absence of mitral diastolic or presystolic murmurs after a test exercise. An auricular septal defect can be excluded by cardiac catheterization which in bilharzial heart disease demonstrates the absence of an auricular shunt and in more advanced cases shows pulmonary hypertension (Soliman 1950. Barbato and his colleagues 1951).

TREATMENT

The aim of treatment is preventive and urinary or intestinal infections should be treated with a full course of antimony compounds such as tartar emetic or Fouadin (stibophen) with a view to preventing pulmonary embolization. In the early stages of lung involvement antimony may be tried with caution as a means of eradicating the primary visceral infection and preventing further ova from reaching the lungs, but there is danger of an acute pulmonary reaction of an allergic nature. Erfan give antimony to 44 cases of schistosomiasis with pulmonary lesions and 4 of the patients with bronchitic or asthmatic symptoms improved, but in other cases fever, urticaria, acute pulmonary congestion and eosinophilia occurred and 1 patient died suddenly (Erfan and his colleagues 1949). Caution is therefore necessary. Heart failure is an absolute contra-indication to antimony treatment because besides the danger of lung reactions it has a direct toxic action on the myocardium. Congestive failure requires the usual measures such as rest in bed, digitalis, mercurial diuretics and a low sodium diet. Once failure has occurred the expectation of life is relatively short.

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CHAPTER 17

DISEASES OF THE LUNG CAUSED BY FLUKES, ROUND-WORMS AND AMOEBAE

Kenneth M A Perry

PARAGONIMIASIS OR *DISTOMATOSIS*

... *Paragonimus westermani* or *Distoma* ... It is ... certain ... , which ... diaphragm ... ally form ... measure ... have

... up to ... sauce ... y and

... second stage produces ... th the parasite and discharged products in the centre ... se to a suppurative lesion with extensive destruction of lung tissue and the formation of ... the middle In the fourth stage there is an ulcerated lesion ... of the previous stages The general

adult fluke may ... spleen, lymph glands and peritoneal cavity

... symptom There ... onstantly becomes ... the cysts reach the ... pain of that condition ... u Ngoch and

Dje-dt (1949) have recorded empyema developing as a result of pleural effusion There is often an inflammatory reaction in the lung when the patient will have all the symptoms of bronchopneumonia Physical examination will only reveal scattered râles throughout the lungs, until there are the signs of pleurisy and pneumonia Diarrhoea may result from abdominal involvement, and Jacksonian epilepsy, headaches, and hemiplegia from cerebral involvement Sometimes there is also enlargement of the lymph glands and ulceration of the skin

Diagnosis

Diagnosis is established by finding ova in the sputum , by the addition of 0 1 per cent sulphuric acid to the sputum the procedure is rendered more simple Charcot-Leyden crystals are often found in the sputum There is usually a well marked eosinophilia The complement fixation test can be helpful and radiographic examination will show the characteristic cystic changes scattered throughout the lungs

The disease runs a chronic course, with periodic attacks of haemoptysis, but the ultimate prognosis is always unsatisfactory owing to the difficulty of eliminating the parasites Where the brain is involved the disease is always fatal

Treatment

Infection can be prevented by not eating crabs in those parts of the world where the disease is endemic All the water in these areas should be boiled and filtered, and bathing should be avoided in contaminated rivers Potassium iodide, 20 grains, three times a day is said to produce beneficial results and improvement has occurred after the intra-tracheal injection of Lipiodol Emetine and tartar emetic help to relieve the cough and sputum , Prontosil has proved of value in some cases However a 15-day course of 40 millilitres (equivalent to 340 milligrams antimony) per day of Fouadin is probably the most effective treatment

ROUND-WORMS INFECTION

Round worms may be divided into the intestinal worms, and the tissue worms The intestinal worms include *Strongyloides*, *Ankylostoma*, *Necator*, *Ascaris* and *Oxyuris* In the first three of these nematodes the larvae penetrate the skin from water or damp earth and find their way into blood vessels, through which they are carried to the right side of the heart and thence to the lungs In the case of *Ascaris* and *Oxyuris* the ova are swallowed in water or food, pass into the intestines where the larvae penetrate the bowel wall and migrate to the lungs From the lungs both groups progress through the trachea, oesophagus and stomach to their natural habitat in the intestine

In their passage through the lungs all these larval worms are capable of blocking the pulmonary arteries and arterioles, and thus causing small pulmonary infarcts which may give rise to symptoms These consist of bouts of fever associated with cough, sometimes with haemoptysis, and shortness of breath Examination of the chest may reveal areas of crepitations Brandt (1949) had recorded a generalized pulmonary fibrosis which he attributed to *Oxyuris* Skiagrams will show patchy transient areas of opacity Blood count in all cases shows a marked eosinophilia (Loffler, Esselier, and Macedo, 1948)

Prophylactic treatment includes the provision of proper sanitation, the wearing of boots and the treatment of carriers The three specific drugs are tetrachlorethylene, carbon tetrachloride, and oil of chenopodium Tetrachlorethylene or carbon tetrachloride, 3 4 millilitres, and 1 millilitre of oil of chenopodium followed by a saline purgative is the best routine treatment Three doses of santonin, 3 grains may be given on alternate days in the treatment of infection by *Ascaris*, and in this condition hexylresorcinol, 1 grain, may be used Gentian violet with an adult dose of 1 grain in an enteric coated capsule thrice daily for 8 days is the best treatment for *Oxyuris* and *Strongyloides* infestation

Filaria bancrofti is the most important of the tissue round worms It is common in India, the West Indies, Porto Rico, Southern China and the Pacific Islands The intermediate host is the mosquito, which sucks the embryos out of the blood at night Development in the mosquito then takes from 10 to 40 days and the mature embryos are then inoculated into man through the proboscis

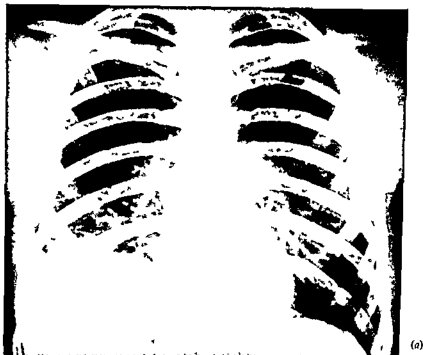
Most of the literature of the disease is concerned with elephantiasis, a complication of filariasis observed in natives after prolonged exposure The acute and early stages of the disease in white man were reviewed by Coggeshall (1946) and consist mainly of lymphangitis, lymphoedema, lymphadenitis, and funiculitis, and are accompanied by aches and pains in the major lymphatic areas Jaffe (1945) found that the lung was in rare instances, involved with the skiagrams showing a patchy type of consolidation with clearing in one part of the lung and development of patches of infiltration in other areas

Hetrazan (1 diethyl-carbamyl-4 methylpiperazine hydrochloride) given by mouth in dosage of 1-2 milligrams per kilo of bodyweight thrice daily for a period of 2-4 weeks is the best treatment Antimony compounds such as Neostibosan and Fouadin have also been used Prophylaxis includes destruction of mosquitos and the use of mosquito nets

THORACIC AMOEBIASIS

Amoebiasis is a disease widely distributed throughout the tropics *Endamoeba histolytica* gains entrance to the body in its cystic form through the mouth in food or water The cysts pass through the stomach, their walls are then digested by the intestinal juices, the amoebae escape and invade the colonic mucosa giving rise to the classical intestinal amoebiasis They may then pass through the portal vein to the liver causing either a diffuse hepatitis or a liver abscess Mostly thoracic amoebiasis is secondary to these conditions but solitary abscesses within the lung substance and not adjacent to the right diaphragm have been described by Dormer and Friedlander (1941) Manson Bahr (1923) and Zaky (1943) The abscesses must be embolic in origin and it is most probable that the amoebae reached the pulmonary circulation by going through the haemorrhoidal veins into the inferior vena cava, or by anastomotic channels between the venules of the portal and vena caval systems within the liver Derrick (1949) reported a fatal case of amoebiasis with lung involvement caused by *Iodamoeba butschlii* that occurred in a Japanese prisoner of war camp There are no other pathogenic amoebae

Pulmonary disease in relation to the right diaphragm is probably much commoner than is generally recognized. The nature and extent of the disease depending on



(a)



(b)

FIG 97 — (a) Amoebic hepatitis showing humping of diaphragm and exaggeration of basal marking (b) showing the humping of the diaphragm in this patient it is posterior

that in the liver These thoracic complications nearly always occur as a direct extension of the abscess or hepatitis through the diaphragm into the pleural cavity, or into the lung through adhesions that have resulted from the inflammatory process Abscesses may form in the lung, or the disease may in fact be the drainage of an hepatic abscess through the bronchus

In at least half the cases of thoracic amoebiasis there will be no history of previous dysentery, though the patient may have had an unexplained fever with pain over the liver from amoebic hepatitis The most usual initial symptoms of the thoracic disease is pain in the lower right chest and in the right shoulder Cough comes next, at first, unproductive, but later giving rise to haemoptysis and the classical "anchovy paste" sputum There will always be fever, sometimes rigors together with anorexia, fatigue, malaise and often extreme loss of weight

Besides this usual manifestation of thoracic amoebiasis lesser pulmonary conditions may result as sympathetic reaction to the liver disease as is stressed by Bookless (1950) These include restricted movement of the right dome diaphragm, serous pleural effusion on the right side, or basal fibrinous pleurisy with a pleural rub, bronchitis, right basal bronchopneumonia and even right lower lobar pneumonia

Empyema is a serious and frequent complication, as may be expected in an infection which crosses the diaphragm to reach the lung Oschner and DeBakey (1943) have recorded a mortality rate of 78 per cent of cases reported in the literature with this complication, as against 40 per cent with a lung abscess

The x ray appearance of the chest in thoracic amoebiasis is characteristic (Fig 97) There is a hump like localized elevation of the diaphragm, which is best seen in the lateral picture Radiating up from this to the hilum will be exaggerated markings made by the track of the infection If a pulmonary abscess develops there will be the usual appearance of an abscess, and similarly with pleural effusion or empyema the picture characteristic of these conditions will appear

Treatment

Emetine is a specific drug for the treatment of thoracic amoebiasis As soon as the diagnosis is made a course of emetine hydrochloride, 1 grain, given intramuscularly daily for 10–12 days, should be instituted Oschner and DeBakey (1943) showed that among 153 patients with this disease who were not treated with emetine 56 6 per cent died, whereas where emetine was used the mortality rate dropped to 1 9 per cent, while in their own personal series the mortality rate of those who did not have emetine was 60 per cent, whereas there were no deaths in those who were treated with this drug

Elsden Dew, Armstrong and Wilmot (1952) have shown that aureomycin and terramycin are effective in the treatment of acute amoebic dysentery, though relapse is not unusual and a state of symptomless cyst passing is apt to develop It is probable that the main action of these drugs is on organisms other than amoebae but there is evidence that *in vitro* they do have some direct anti amoebic effect There is therefore strong reasons for using these two drugs in conjunction with the established amoebicides

Surgical intervention, as is pointed out by Shaw (1949) is needed only when secondary infection has occurred, when there is an empyema which cannot be cured

by repeated aspirations, when a persistent abnormal channel between the bile duct system and bronchi persists, or when fibrosis of the pulmonary tissue continues to produce symptoms as a result of the abscess The operations which will most frequently be required are, drainage of empyema or liver abscess in the latter case the mortality will be greatly reduced by using the extra-serous route, or lobectomy or thoractomy when there is damage to the lung

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CHAPTER 18

HYDATID DISEASE OF THE LUNG

GEOFFREY FLAVELL

AETIOLOGY

THE DEFINITIVE host of *Taenia echinococcus* is the dog, in whose intestine the worm of 0·5 centimetre length lives, the intermediate hosts are sheep, pigs or men, in whom it undergoes its larval and encysted forms Dogs become infested by eating sheep's offal, and man by direct contact with dogs or from eating lettuce and other produce contaminated by their faeces In Argentina, Australia, New Zealand, the Middle East, Iceland, and some parts of Wales the disease is common, elsewhere it is rare The liver is the favoured site of cysts, accounting for 75 per cent of the cases, but, according to experienced Australian surgeons, up to 25 per cent are found in the lung

After the ova have been discharged from the gravid terminal segment of a worm, and have found their way into the human stomach, gastric juices digest their outer capsule freeing an embryo, or hexacanth, from its six thorns, which migrates through the stomach wall, enters the portal vein, and so reaches the liver Should it pass this filter it finds itself in the inferior vena cava, and so passes *via* the right heart into the lung, where it lodges, and enters a larvated stage The embryo vacuolates and forms a small vesicle composed of two walls an inner nucleated germinal layer and an outer non-nucleated laminated layer This now expands into a cyst which is small at first, but, if it is not disturbed, becomes progressively larger until it may reach a considerable size From the germinal layer bud off groups of cells that in turn vacuolate to form proliferative " brood capsules " giving rise to scolices, the heads and source of new worms Most of these become detached, and sink through the hydatid fluid, forming " hydatid sand " upon the floor of the cyst, while outside the laminated layer the lung reacts to its presence by condensing an adventitious layer of fibrous tissue about it, but easily separated from it, as is the skin from a banana

CLINICAL PICTURE AND DIAGNOSIS

The fate of the cyst, and the symptoms of the patient, now depend upon the complications which may arise During the period of growth the cyst gives no

fixation test is now seldom used As the cyst increases in size pain may occur, together with pressure symptoms, and occasionally there may be anaphylaxis with

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CLINICAL PICTURE AND DIAGNOSIS

The fate of the cyst, and the symptoms of the patient, now depend upon the complications which may arise During the period of growth the cyst gives no

at this stage is by the intra-dermal Casoni reaction, which is not always reliable, or the existence of an eosinophilia of 4 per cent or more The Weinberg complement fixation test is now seldom used As the cyst increases in size pain may occur, together with pressure symptoms, and occasionally there may be anaphylaxis with

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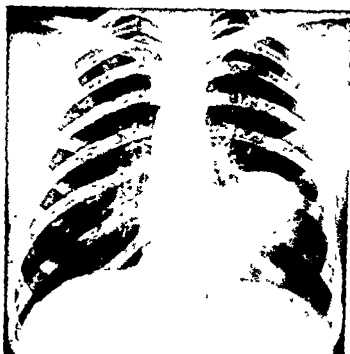


FIG 98 —(*a* and *b*) Large hydatid cyst situated in lower lobe of left lung in a young soldier who had served for a short time in Greece where he had kept a pet dog A Casoni test before operation was negative, and from its position and appearance the x ray opacity was thought to be caused by a neurofibroma Successful removal at operation, with no recurrence

(*a*)



(*b*)

severe bronchospasm dyspnoea and tachycardia though this is more often seen during complications or after surgical intervention

The zone of atelectatic and fibrous tissue of the adventitious layer tends gradually to cause pressure erosion as it comes to abut upon a bronchial wall and finally a fistula is established Air escapes into the adventitious pericyst stripping the true hydatid from it If the cyst is small it may now be expectorated *in toto* and the patient be spontaneously cured In many instances rupture of the cyst occurs and hooklets scolices or fragments of laminated membrane may be expectorated and confirm the diagnosis The cyst does not necessarily become infected at this juncture though it may do so but either dies or continues to live and perhaps to produce daughter cysts

The intrusion of air into the pericyst with separation of the hydatid sometimes gives rise to x ray appearances which Argentinian and French writers have described as perivesicular pneuma the double dome arch or Cumbo s sign and camalote formation according to the amount of air between the pericyst and the cyst itself since this air shows as a crescentic translucent area between the two opaque convexities of the cysts Unfortunately these so called pathognomonic signs are neither reliable nor common

If the cyst ruptures into a bronchus or the pleura scolices are spread and multiple cysts develop simultaneous bronchial and pleural fistulae result additionally in pneumothorax (Fig 99) Metastatic hydatidosis follows erosion and rupture into a pulmonary vessel Multiple cysts of this kind occur in about 6 per cent of infestations and are commoner in children rupture itself carries a heavy mortality

Once a fistula has been established ultimate infection will probably follow bringing with it purulent sputum fever haemoptysis and general evidence of pulmonary suppuration If a hydatid cyst has obstructed a bronchus however collapse and secondary bronchiectasis may result with similar symptoms Infection also adds to the risk of pleural rupture since empyema will then accompany dissemination Sometimes the cyst dies after it has become infected but daughter cysts can continue living even amongst pus They are more commonly associated with hydatid cysts of the liver however and should they appear in the sputum hepatic involvement must be feared for liver cysts when infected can perforate the diaphragm and rupture into the lung the pleura or the mediastinum Infestation of more than one organ occurs in less than 2 per cent of the total number of cases

Hydatid cysts seldom die except as a result of rupture or infection and even calcification of the cyst does not necessarily imply the death of the parasite If a cyst is removed or is coughed up the pericyst is usually soon obliterated by re expansion of the lung but should it be unusually rigid may persist and in the course of time become epithelialized by extension from the bronchial mucosa and form a permanent lung cyst which in its turn may become the seat of infection

TREATMENT

The treatment of hydatid cysts of the lung is surgical and has for its object the complete removal of the parasite with maximum conservation of pulmonary tissue Expectant treatment (except perhaps in the case of very small cysts detected



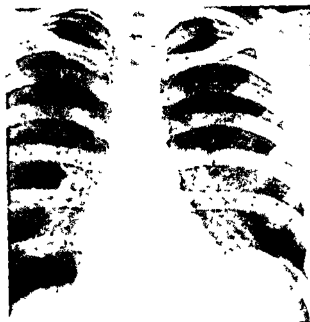
FIG 99 — (*a*) Large hydatid cyst in left lower lobe of a school boy evacuated to Wales during World War II While awaiting admission to hospital he played a game of football and the cyst ruptured into the pleural cavity producing a hydropneumothorax, (*b*) thick cyst wall can now be seen Moderate anaphylaxis developed At operation the cyst was found to be heavily infected and a bronchial fistula was also present, (*c*) left lower lobectomy with complete re-expansion of remaining lung with no recurrence

(*a*)





(*b*)



(c)

by routine radiography), transpleural aspiration, the induction of an artificial pneumothorax, and even bronchoscopy are contra-indicated since all may favour rupture with dissemination After careful radiographic localization the chest is opened where access to the cyst can best be obtained Pleural adhesions cannot be relied upon securely to isolate the rest of the pleural cavity, nor does it seem profitable to try and secure adherence by packing the extrapleural space or sewing visceral to parietal pleurae, and removing the cyst at a later stage The pleural space is carefully packed off, some surgeons employing black towels the better to detect contamination, and the cyst fluid is partially and slowly aspirated with a fine-gauge needle attached to a two-way syringe A small incision is then made down to the pericyst and the glistening white hydatid is removed from it complete, meticulous care being taken to avoid rupture or the spilling of any of the contents The interior of the pericyst is then inspected, bleeding points are secured, and any fistula that may exist closed The walls of the pericyst itself may be approximated, though it usually soon obliterates Multiple cysts are dealt with similarly

Should serious bleeding occur, as it sometimes does, or if a cyst be very large, or accompanied by bronchiectasis, lobectomy is performed When the cyst has already ruptured, and is infected, lobectomy may likewise be necessary , however, every effort should be made to conserve the lung, and if local removal of the cyst can be achieved without further contamination it is to be preferred, both pericyst and pleura being drained

Barrett (1949) has pointed out that hydatid cysts of the lung show a natural tendency to herniate from the lung as they increase in size, and has recently devised a technique of removal which profits by this, and delivers the cyst intact avoiding even preliminary aspiration which with the finest needle may sometimes lead to a tear of the laminated membrane With patient's head lowered, so that if rupture occurs into a bronchus natural drainage takes place, and with closed circuit anaesthesia, the chest is widely opened and the cyst identified A macintosh bag, with strings round its neck and its bottom open, is then placed over the affected lobe and the strings drawn just tightly enough about the hilum not to obstruct the circulation The open end is brought out of the wound and spread on the surface so that the part is completely isolated within its bag The adventitia is then incised until the bluish-white laminated membrane is just exposed at one point, and freed gently for about an inch The anaesthetist now softly increases the pressure in his circuit, and little by little over a period of 10–30 minutes, the adventitia cracks along the line of the incision and the cyst gently extrudes, to fall at last intact into the protecting bag

Many hydatid cysts of the heart have been described, and are said to occur in some 2 per cent of infestations Occasionally recognized radiologically, they are associated at times with paroxysmal tachycardia, and often terminate fatally by rupture which may result in embolism from the liberated daughter cysts d'Abreu (1950) has described his successful removal of such a cyst from the wall of the left ventricle employing the usual technique, with preliminary aspiration, for hydatids of the lung and it seems probable that his lead will be followed by increasing numbers of direct attacks upon this hitherto inviolate refuge of the parasites

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CHAPTER 19

SYPHILIS OF THE LUNG

KENNETH M A PERRY

SYPHILIS of the lung is a very rare condition It may be either congenital or acquired The congenital form is the commoner and is usually found in stillborn syphilitic infants It has been called pneumonia alba The gross appearance of the lung is characteristic, the lung being of a pale greyish colour and consolidated throughout, with the essential portions sinking in water, and the surface dry and smooth The histology is that of arrested development rather than active inflammation Either the alveoli have failed to develop or they are smaller than normal and lined with cubical epithelium

In adults, gummas may develop in the lungs, they may be either single or multiple, when they are multiple they are usually small and do not give rise to symptoms.

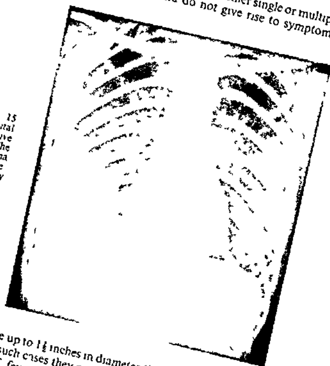


FIG 100—A girl aged 15 years with congenital syphilis and a positive Wassermann reaction The radiograph shows a gumma in the right lower lobe which was subsequently proved histologically

Large gummas, however, may be up to 1½ inches in diameter (Fig 100) They sometimes undergo softening and in such cases they may present as a lung abscess The patient may then complain of fever, cough and persistent purulent sputum



(a)



(b)

FIG 101 —(a) Female aged 35 years with swelling over manubrium and rales at left base Wassermann reaction positive (b) 3 months later after treatment with potassium iodide 25 grains 3 times a day and intramuscular injections of bismuth

Examination of the chest may show diminished air entry and rales in the area affected The process may progress and give rise to lesions which are similar to bronchiectasis or fibro caseous tuberculosis

DIAGNOSIS

The diagnosis is always difficult but the Wassermann reaction will be positive Skiagrams of the chest show either a solid opacity or a cavity Tubercle bacilli will be persistently absent from the sputum The diagnosis may only be proved when an excised portion of the lung is examined histologically

TREATMENT

If the lesion is not soft and broken down it may respond satisfactorily to anti syphilitic treatment Arsenic bismuth mercury and penicillin are the drugs used to destroy the spirochaetes but iodides are valuable in stimulating the removal of granulomatous tissue (Fig 101) The following is satisfactory treatment On the first day the patient is given an intravenous injection of 0 3 gramme bismuth oxychloride phenamine and an intramuscular injection of 0 45 gramme neoars followed on the next five days with intramuscular injections of 1 million units of procaine penicillin On the seventh day 0 6 gramme of neoarsphenamine and 0 3 gramme of bismuth oxychloride and on the following five days 1 million units of procaine penicillin are administered This should be followed by six weekly doses of 0 6 gramme neoarsphenamine and 0 3 gramme bismuth oxychloride A second and third course may be given if necessary If however the lesion has broken down to form cavities and lung tissue has been destroyed the only method of treatment is by excision of the affected part of the lung Any attempt at drainage will be followed by permanent sinus formation

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CHAPTER 20

PULMONARY EMBOLISM

W D W BROOKS

AETIOLOGY AND INCIDENCE

PULMONARY embolism is not a new disease but in recent
become increasingly aware tha
for a variety of reasons new in
and treatment It may be defi
tion as a result of abnormal st

Many different kinds of emboli may thus reach the lung For example, air may enter the jugular vein when the throat is cut and
circulation in the heart and pulmonary
in the blood and so pulmonary emboli
embarrassment when rapid decompres
rapidly to the surface The larval stage of some parasites may cause pulmonary embolism, for instance, in ankylostomiasis Bony fracture or penetrating wounds involving fatty tissues, or the use of mineral oil as a lubricant when passing a catheter may admit large amounts of fat or oil to the circulation and so cause

may derive from infected thrombi in cases of septic abortion, osteomyelitis, and in the course of pyaemia or septicaemia, while perhaps the commonest bacterial embolism may occur in the early months following primary tuberculous infection Many of these emboli may give rise to specific effects within the lungs dependent upon their nature, and miliary tuberculosis, multiple staphylococcal lung abscesses or metastatic carcinomas illustrate their variety These features are dealt with elsewhere, moreover, their aggregate is small in comparison with the common form of pulmonary embolism which arises as a result of the detachment of a clot or clots from a preformed venous or cardiac thrombosis We are concerned here with this last and most frequent form; the special types mentioned above will be considered only in so far as they contribute to the clinical features common to all forms of pulmonary embolism

that is within the hour, for at least 60 per cent of patients with fatal emboli survive longer, some for several days Clinically diagnosed pulmonary embolism occurs in perhaps 1 per cent of all hospital patients, while certainly many more cases are undiagnosed or are thought to be instances of pneumonia, massive collapse of the lung, or other disorder

The primary source within the venous system is often not obvious It is not sufficiently recognized that in apparently healthy adults the chance that the plantar or calf veins carry thrombi is about evens that skiagrams of the pelvis commonly show phleboliths and that it is uncommon not to find thrombosed veins in the broad ligament at hysterectomy As Virchow showed the great majority of emboli arise from thrombi in the veins in these areas—the legs and the pelvis All other areas including the right side of the heart are relatively uncommon sources Ochsner has distinguished between thrombophlebitis due to local trauma or bacterial invasion with a relatively firm adherent clot and well marked local symptoms and signs and phlebothrombosis due to venous stasis and alterations in the cellular and fluid constituents of the blood which increase its coagulability with a loosely attached clot and few if any local clinical manifestations In the former unless there is suppuration embolism from the thrombus is rare in the latter it is common Conditions which predispose to phlebothrombosis include local venous obstruction immobilization and heart failure post operative and puerperal states specific fevers and polycythaemia The incidence of venous thrombosis increases with age and in cold seasons and climates

PATHOLOGY

As a rule the obstruction of one or a few minor branches of the pulmonary artery by an embolus or emboli has no demonstrable effect upon the lung So efficient is the blood supply *via* the bronchial arteries that major or many minor branches of the pulmonary artery may be obstructed without infarction of the lung occurring Such thrombi contract become organized and recanalization with restoration of the circulation takes place However when the circulation from the bronchial arteries is defective for example in heart failure or arteriosclerosis embolism more easily causes pulmonary infarction The infarcted area of lung is at first swollen hard dry and bloody in colour The adjacent pleura is roughened with a layer of fibrin and later quite a considerable blood stained effusion may develop As a rule the embolus can be found to be obstructing the pulmonary artery concerned Microscopically the area of lung involved is grossly distended with coagulated blood and in the course of time death reabsorption of the endothelial cells lining the alveoli and coagulation necrosis occurs It is followed by much contraction of the aging infarct and replacement with fibrous tissue A further consequence of pulmonary embolism acute cor pulmonale does not develop unless about two-thirds of the pulmonary circulation is obstructed Thus a rider embolus at the bifurcation of the pulmonary artery or numerous simultaneous or successive emboli in each lung may provoke sufficient rise of pressure proximally to cause acute right ventricular failure which may well be fatal Infarction of the lung occurs variably in such cases Apart from this cause of death which may be rapid massive pulmonary embolism may kill speedily from shock asphyxia and insufficient circulation of blood to the left heart It is thought also that widespread vagal stimulation is provoked by pulmonary embolism affecting the heart pulmonary vascular tree bronchi and gastrointestinal tract Such autonomic effects augment the above mentioned consequences of massive embolism and may well explain the occasional fatalities



FIG 102 — Multiple sarcomatous emboli—primary in tibia—with small bilateral effusions

following apparently minor emboli Air, fat, or oil may cause either simple embolism, infarction or acute cor pulmonale, and death, while recurrent embolism especially with *carcinomatous emboli occasionally gives rise to relatively slowly* developing right ventricular failure during the course of which the sequelae of widespread pulmonary neoplastic metastases can develop (*Fig 102*) *Finally,* it must be remembered that minor and many major consequences of pulmonary embolism may be produced by thrombosis occurring in the pulmonary arterial tree itself, and that this is especially liable to occur when the pulmonary circulation is obstructed in heart failure or mitral stenosis There is some evidence *that digitalis is a significant predisposing factor to thrombosis in* pulmonary and systemic vessels

SYMPTOMS AND SIGNS

Pulmonary embolism commonly occurs without symptoms or signs of any kind, and it is equally true that other features of the case when *heart failure is* present for example, may so obscure the clinical picture as to make diagnosis almost impossible Quite often restlessness, fear, or vague uneasiness may be ... ght increase ... te will be ... is referable ... n the legs,

but it is uncommon to find the classical signs there of thrombophlebitis because when oedema, pain, a tender hard vein, and local discoloration are present the thrombus is firmly adherent The really dangerous phlebothrombosis is relatively silent but may cause slight swelling of the lower leg, and since it is usually situated in the deep veins the more superficial are often distended Local tenderness, if carefully sought, may be found in the sole of the foot near the external malleolus, or in the calf, and dorsiflexion of the foot may cause pain in the same site The temperature of the affected leg may be raised

Variable local features may be associated with pulmonary embolism For example, many cases confidently diagnosed as massive collapse or broncho-pneumonia following operation or childbirth are really examples of embolism The signs and symptoms in such cases may closely resemble those of the disorders they mimic

However, while pulmonary embolism with or without infarction often has no characteristic clinical features within the chest, it is also true that quite clearly recognizable symptoms and signs may be produced there (Fig 103) An abrupt onset with pleuritic pain, cough haemoptysis often lasting for some days, and cyanosis and dyspnoea may occur These may be associated with a pleural rub, signs of consolidation, persistent local adventitious sounds, fever, tachycardia, and later transient haemolytic jaundice It is not uncommon, when infarction of the lung has resulted, for an appreciable blood-stained pleural effusion to develop and to mask other signs Since embolism is often multiple the signs frequently involve both lungs, moreover, successive episodes may take place [illegible]

and air entry, and an impaired percussion note Compensatory emphysema is associated, but it is unusual, save with infected emboli, for bronchiectasis to develop Only large infarcts are likely to leave behind recognizable permanent signs such as these, and the patient thus damaged is likely to be easily dyspnoeic thereafter

The characteristic and dominant feature of pulmonary embolism of a considerable order of magnitude is its striking catastrophic onset Scarcely less significant is the association of symptoms and signs of shock, namely faintness, pallor, sweating a low blood pressure, vomiting, and tachycardia with those mentioned above marking the arrival of the embolus within the pulmonary arterial tree

When massive embolism occurs death may come swiftly by shock, virtual cessation of the circulation and asphyxia With rather more protracted survival, and if more than about 60 per cent of the circulation is obstructed the syndrome known today as acute cor pulmonale may develop As the embolism occurs the patient is seized with a sense of [illegible]
sust [illegible]
and [illegible]
As a rule there are no abnormal signs to be heard over the lungs (Fig 104) The cardiac signs are those of rapidly developing dilatation right ventricular failure, and may include marked pulsation in the second and third interspaces



FIG 103 — Recent large infarct right lower lobe with an associated pleural effusion and slight *mediastinal shift to the right* This occurred on the tenth post-operative day after partial *gastrectomy*

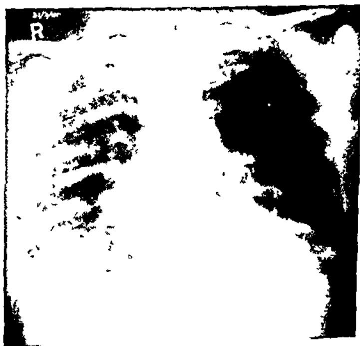


FIG 104 — *Multiple pulmonary emboli* producing small infarcts with *acute pulmonary hypertension and right ventricular* strain

to the left of the sternum, protodiastolic gallop rhythm best heard in the same site, accentuation of the pulmonic second sound, a pericardial rub in this same area, and venous distension in the neck, and of the liver The blood pressure falls markedly The electrocardiogram is helpful in the difficult distinction from coronary infarction in a minority of cases When this is so the main characteristics in acute cor pulmonale are —

(1) A prominent S in lead I;
(2) T is low or inverted in lead II,
(3) In lead III a Q wave develops or is increased, and the S T segment may be elevated, T is deeply inverted
(4) In lead IV (R or F) T is flat or inverted, while T inversion is common in C R I

Radiological appearances

Correlation between radiological findings and the clinical severity of the case is not marked Serious and repeated embolism may for instance occur and the chest be normal radiologically Almost any type of parenchymal shadow may be observed if infarction occurs, the commonest being a peripherally situated irregular area of consolidation with an associated pleural reaction The classical wedge-shaped area extending out from the hilum is rarely seen Elevation of the diaphragm on the affected side, pleural effusion, and atelectasis often complicate embolism, while linear and more extensive scarring may remain permanently when the patient recovers

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Pulmonary embolism should be suspected when the appropriate clinical antecedent conditions exist if otherwise unexplained fever, dyspnoea, and tachycardia develop Evidence of phlebothrombosis will increase the probability of the diagnosis When an abrupt worsening of the patient's condition occurs accompanied by signs and symptoms referable to the chest of the type mentioned diagnosis is as a rule easy, particularly if shock is associated In these cases radiology is often helpful Massive collapse of the lung also occurs abruptly but usually has characteristic physical signs, in uncomplicated cases does not quickly give rise to haemoptysis, tends to follow sooner after operation, and the radiological findings are frequently diagnostic Broncho-pneumonia is of much slower development, the sputum is purulent, the signs even more often bilateral Coronary thrombosis may closely resemble acute cor pulmonale, but the pain is commonly more severe, pulmonary congestion and orthopnoea are often present, restlessness greater, and the electrocardiogram as a rule abnormal and different from that set forth above

TREATMENT

The prevention of thrombosis in the veins of the legs and pelvis is important Many surgical and perhaps most medical patients could with advantage have the circulation in these areas improved by repeated breathing exercises in conjunction with massage and leg exercises, and so decrease the risk of thrombosis In

hospital many patients are unnecessarily immobilized in bed The provision of a dining hall and recreation room would do much to reduce this and incidentally the prevalence of pulmonary embolism Finally, the proper treatment or prevention of anaemia, dehydration, and haemoconcentration are also significant prophylactic measures

If phlebothrombosis is strongly suspected or known to be present anticoagulant therapy is desirable Heparin is given at 4 hourly intervals in 50-milligram doses undiluted intravenously Simultaneously, oral administration of dicoumarol is started using 300 milligrams on the first, and 200 milligrams on the second day

The blood prothrombin levels must be determined on alternative days, and the dosage of dicoumarol is adjusted on subsequent days to keep this level between 30 and 60 per cent of normal Heparin is discontinued after 2 days, but, if it is essential, the dicoumarol may be continued for 1 or more months The drug should not be discontinued abruptly, but should be reduced slowly over the period of a week Breathing exercises at this stage are contra indicated Tromexan may be used as an alternative to dicoumarol Its action is quicker, and it is less dangerous However, great variability is shown by different patients, and by the same patient from time to time, to its action Marked drug resistance is quite commonly a most irritating disadvantage

A loading dose of 1,200 milligrams, in an urgent case accompanied by 100 milligrams of heparin given intravenously, is followed next day by 900 milligrams, and thereafter 300 milligrams daily are given by mouth, provided estimates show that this dosage is adequate to give rise to and maintain a prothrombin time between 10–30 per cent of the normal

Anticoagulant therapy may also of course be used after embolism has occurred, with the object of preventing further formation of thrombus and so diminishing the risk of recurrent embolism which may well be much more severe Contra-indications to the use of this treatment include severe kidney and liver disease, blood dyscrasias, recent operation on the brain and spinal cord, high fever, and bacterial endocarditis

The method of treatment is not without danger and, since fatal haemorrhage may occur, the above mentioned method of control is essential Even so it is urged that if any spontaneous haemorrhage however slight occurs, or if the patient complains of backache dicoumarol administration should be stopped [illegible] haemorrhage for [illegible]

Surgical measures have been devised to prevent emboli reaching [illegible] They include proximal vein ligation, for example the femoral vein if the calf veins are the site of phlebothrombosis, or if both legs are involved ligation of the inferior vena cava These methods are often combined with anticoagulant therapy While the proper limitations of this method of treatment are not yet settled there exists a measure of agreement that it is of value (1) after unexpected pulmonary embolism, for which good evidence of the source exists, (2) when phlebothrombosis develops and the patient is likely to be immobilized for some other cause, and (3) to prevent spread of the clotting process

When massive pulmonary embolism occurs atropine sulphate $\frac{1}{50}$ grain, and $\frac{1}{2}$–1 grain of papaverine hydrochloride should be given intravenously at once Oxygen should be administered, and anticoagulant therapy started Because morphine is alleged to increase the coagulability of the blood pethidine or physeptone are preferable for the relief of pain, while papaverine is more effective in preventing vagal effects Pulmonary embolectomy has a great mortality, and should only be used in those patients surviving the immediate risk for whom early death seems otherwise inevitable Venesection, digitalis, and vasodilator drugs are valueless Intravenous strophanthin 0 5 milligram followed by 0 25 milligram every 8 hours for 2 days may help increase the cardiac output About two thirds of the cases of acute cor pulmonale survive, and for these after the emergency is over good nursing anticoagulants and possibly venous ligation are essential

When pulmonary infarction occurs, depending on the severity of the case measures selected from those mentioned above will be needed In addition pleuritic pain should be relieved by strapping and an electrically heated pad Expectoration should be encouraged In some cases, at a later stage iron by mouth and liver injections may be required if a significant anaemia has developed

PROGNOSIS

Recovery is finally complete in a majority of cases of pulmonary embolism At the onset however, it is rarely possible to give any indication at all from the available clinical features of the fate of a particular case The risk of repeated emboli should be remembered in even the mildest case In general however, the more severe the early symptoms and signs, the graver is the prognosis

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CHAPTER 21

BLAST INJURIES OF THE LUNG

KENNETH M A PERRY

AETIOLOGY

ZUCKERMAN (1940) showed that in animals exposed to blast, haemorrhages occurred in the lungs These could consist in (1) a few small haemorrhages on the surface (2) patchy haemorrhages scattered throughout the lungs or (3) complete hepatization of the lungs by haemorrhages He thought that the lesions were produced by the wave of positive pressure acting directly on the chest wall, and this theory is generally accepted

CLINICAL FEATURES

Condition immediately after exposure

The patient is frequently unconscious for a short time at the time of the injury, when he comes round he is usually quiet and calm, in contrast with the anxiety of patients who have large haemorrhages The most striking symptom is shortness of breath, which may last for several days after the injury Breathing is sometimes painful, and the patient usually complains of a feeling of tightness in the chest Apart from pain on breathing, the patients sometimes complain of retrosternal pain, or pain in the intercostal muscles These may remain tender for a week or more Most patients develop a cough and expectorate frothy (usually blood stained) sputum for several hours after the injury After this the cough often becomes dry The patients sometimes complain of epistaxis and deafness

Condition on examination in hospital

Temperature, pulse and respiratory rates are usually normal by the time that the patient is admitted to hospital In the chest, dullness, diminished breath sounds and decreased fremitus are usually present Generalized rhonchi are found in most cases and persist for a few days The radiological appearances are variable and there is nothing specific about them They may be unilateral or bilateral [illegible] found to be

the patients usually recover

TREATMENT

Treatment is essentially symptomatic, rest being the most important single requirement, but the areas of haemorrhage sometimes become infected and pneumonia

may develop This should be treated by chemotherapy Physiotherapy is indicated for the muscular pain which is so prominent a symptom Breathing exercises are also of great value

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CHAPTER 22

PULMONARY HAEMOSIDEROSIS

KENNETH M A PERRY

AETIOLOGY

PULMONARY haemosiderosis is described in two associations, firstly as an *idiopathic* disease, and secondly in conjunction with mitral stenosis Idiopathic haemosiderosis is a rare condition which occurs mostly in children under the age of 16 years Borsos-Nachtnebel (1942) described the only case in the literature in an adult; the patient was a man aged 38 years Ceelen (1931) was the first to describe the condition Wyllie and his colleagues (1948) collected 17 cases from the literature and described 7 of their own, and further cases have been reported by Waldenstrom (1948) Nancekievill (1949), King (1949), and Walton and Williams (1951)

IDIOPATHIC PULMONARY HAEMOSIDEROSIS

Clinical picture and diagnosis

Symptoms consist in recurrent attacks of fatigue, pallor, cyanosis and increasing dyspnoea and palpitation These symptoms may be of sudden onset There is usually a cough, which is troublesome and often associated with vomiting Haemoptysis is frequently present There is sometimes severe abdominal pain and the patient becomes restless and apprehensive Each attack commonly lasts [illegible] the more acute cases, [illegible]

failure resulting from a severe disturbance of the pulmonary circulation This is well shown b [illegible] ormal and show a diffuse, extre [illegible] (*see* Fig 105) [illegible] more accentuated during an acute attack, when there may be evidence of a partial lobar or lobular collapse The mottling is usually much finer than that seen in miliary tuberculosis or sarcoidosis The speckling is also smaller than is seen in occupational siderosis

Necropsy findings

At necropsy the main changes are to be found in the lungs Macroscopically these appear full and firm, of a dark reddish brown colour, with fine haemorrhages visible on the pleural surfaces On section the lungs appear solid Enlargement of the broncho-pulmonary and tracheo-bronchial lymph-nodes is conspicuous The right side of the heart is usually increased in size

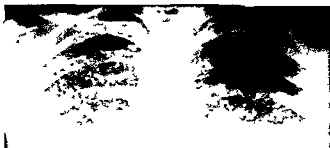
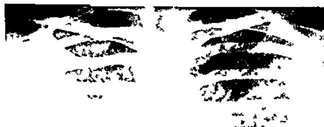


FIG 105 — Radiograph of (*a*) brother and (*b*) sister showing idiopathic pulmonary haemosiderosis

(*a*)



Microscopically, the alveoli are filled with haemosiderin-laden *phagocytes* The epithelial cells of the alveolar walls also contain haemosiderin, and many are desquamated within the alveoli, which also contain red blood cells and haemosiderin granules lying free Haemosiderin granules are also found in the interstitial tissues and the lymph nodes Diffuse pulmonary fibrosis, capillary fibrosis and fragmentation of elastic fibres in the alveolar walls, deposits of haemosiderin from previous haemorrhages and fresh bleeding into the alveoli are also sometimes found

Course and prognosis

The cause of the condition is obscure The prognosis is bad, and there is no treatment which appears to alter the course of the disease

HAEMOSIDEROSIS ASSOCIATED WITH MITRAL STENOSIS

Pathogenesis

It had long been known that the lungs of patients with mitral stenosis show brown induration, but it was not until 1928 that Rosenhagen drew attention to the densely mottled skiagrams of the chest which are sometimes seen in patients with mitral stenosis He showed that these were caused by nodulation and were microscopically multiple focal deposits of haemosiderin Since then the condition has been fully described in America by Soma Weiss (1942) and Pendergrass, Lame and Ostrum (1949), and in Great Britain by Lendrum, Scott and Park (1950) Why the condition occurs in certain cases of mitral stenosis and not in others is not clear Pendergrass and Leopold (1945) suggest that the deposit might be the result of perivascular interstitial haemorrhages, which they believed to occur at the acute phase of the rheumatic disease Other writers still accept pulmonary congestion as an adequate explanation of the skiagraphic nodulation, but Lendrum, Scott and Park (1950) believe that the foci of the haemosiderin are situated in groups of adjacent alveoli and that the distribution is related to the wall of the terminal bronchiole, where the capillary anastomoses between bronchial and pulmonary arteries are believed to lie At this site frequently repeated haemorrhages of mechanical origin occur These are associated with haemoptysis The ferrous impregnation of elastic tissue and reticulin around the haemosiderin causes the breakdown of these structures, with resulting fibrosis This obstructs the lymphatic withdrawal and so perpetuates and intensifies the dangerous concentration of intra alveolar haemosiderin, and in some of these cases an osseous mass develops

Necropsy findings

Histologically the only difference between the two varieties of pulmonary [illegible] iron is a focal [illegible] iopathic

Diagnosis

Skiagraphic appearances show the characteristic cardiac shadow of mitral stenosis There are fine nodular opacities in the lungs, which are most numerous

in the mid zones and densest at the hila they produce a clouded or marbled effect Towards the periphery and the bases they are fewer in number, and the apices may be clear

Prognosis and treatment

Haemosiderosis does not appear to affect the prognosis of mitral stenosis Treatment should be directed to the treatment of the cardiac lesion, there is no specific treatment for the pulmonary disease

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Idiopathic Haemosiderosis

CHAPTER 23

OCCUPATIONAL LUNG DISEASES

KENNETH M A PERRY

IT HAS been known since prehistoric times that dust will produce diseases of the lung, and conditions of this type were termed pneumonoconiosis (Greek *pneumon* — lung, *konis* — dust) *The term has since been incorrectly corrupted* in England to pneumoconiosis, which translated literally means ' air dust " The term is an all inclusive one but is usually adapted to refer to dusts which are either inert, such as iron, or give rise to a fibrosis of the lung as do dusts like silica, asbestos, certain other silicates and coal The dust must be of a particle size of less than 5 μ to pass through the smallest bronchioles and thus reach the alveoli and so give rise to pathological lesions Besides these dusts certain metallic dusts such as manganese, vanadium, cadmium osmium and beryllium give rise to either acute or chronic pulmonary oedema or pneumonia Pollens and dusts of [illegible] asthma which is described else where
the con [illegible]
produce a characteristic asthma which will be referred to below Organic dusts *arising from hay, straw, grain, bagasse, and cotton produce a group of diseases* which have been labelled ' asthma ", " bronchitis ", and " pneumonia " These sometimes resolve leaving little disability but in other cases run a protracted course with fibrosis of the lung and bronchiectasis

PLATINUM ASTHMA

As long ago as 1911 Karasek reported that workers in photographic studios in Chicago suffered from irritation of the throat and nasal passages, which caused violent sneezing and coughing The bronchial irritation was sufficient to cause *marked shortness of breath and a wheeze It was found that the cause* of this condition was the use of paper containing potassium chloro platinate, and when this paper was no longer used the symptoms subsided Refining of platinum involves the production of the complex salts of platinum and at least half of the workers in this industry suffer from a syndrome similar to that produced by the photographic paper The symptoms are repeated sneezing followed by profuse running of the nose with watery discharge, This is followed by tightness of the chest shortness of breath and a wheeze The symptoms persist as long as the worker remains in the factory and for about half an hour after he has left they then subside However the patient may wake in the early hours of the morning [illegible]

TABLE I

DEATHS FROM FIBROSIS OF THE LUNG INCLUDING SILICOSIS ASBESTOSIS PNEUMOCONIOSIS AND BYSSINOSIS (ENGLAND AND WALES) 1940–1949
(From the Annual Report of Chief Inspector of Factories 1949)

	1940	1941	1942	1943	1944	1945	1946	1947	1948	1949
Silicosis										
Refractories industries	16	12	7	7	6	9	8	10	13	13
Pottery manufacture of	53	45	47	41	32	41	49	54	48	63
Sandstone quarrying and dressing	42	24	14	24	26	21	20	12	28	15
Sandstone masons	67	58	40	53	31	44	41	43	40	36
Metal grinding	24	13	13	11	7	11	6	9	8	14
Sandblasting	13	4	4	6	7	6	4	5	5	5
Steel dressing and cleaning of castings	4	9	9	4	10	5	22	13	13	18
Stone pebble flint and sand crushing	5	1	2	1	4	—	—	3	1	3
Scouring powders manufacture of	—	2	—	1	1	1	—	—	—	1
Abrasive wheel manufacture	—	—	—	1	1	2	1	—	—	—
Glass cutting and bevelling	—	1	2	—	—	—	2	—	—	—
Millstone dressing	—	—	1	—	—	—	—	—	—	—
Slate quarrying and dressing	7	6	4	6	7	6	13	20	16	21
Granite quarrying and dressing	1	1	3	2	—	1	—	4	1	1
Tunnel mining (sewage works etc)	2	—	5	4	4	4	5	—	3	2
Coalmining	232	196	230	276	277	323	343	347	322	336
Goldmining (South Africa)	10	7	7	4	9	4	7	5	4	4
Tin mining	21	17	12	14	11	19	14	12	11	20
Iron ore (haematite) mining	20	4	7	5	7	2	9	6	12	9
Lead mining	—	1	3	1	1	1	1	2	—	1
Copper mining	—	1	2	1	—	—	—	—	—	1
Barytes mining	3	3	—	4	—	2	1	1	—	3
Clay mining	—	—	—	1	—	1	—	—	—	1
Mining engineers	—	—	3	1	—	—	1	1	2	1
Miscellaneous	2	1	2	2	4	5	6	11	7	7
Total—Silicosis	522	406	417	470	445	508	553	558	534	575
Asbestosis	11	17	11	8	10	11	16	15	15	17
Pneumoconiosis										
Coalmining	—	—	—	—	34	64	78	230	317	420
Other industries	—	—	—	—	3	11	5	12	19	14
Byssinosis	—	—	6	7	1	10	3	4	8	7
Other cases of (non-occupational) Fibrosis	650	461	443	493	531	529	568	616	651	554
Grand Total	1 183	884	877	978	1 024	1 133	1 223	1 435	1 544	1 587

dust of the complex salts of platinum, in a concentration of 5 micrograms per cubic metre or more The disease can be prevented by adequate exhaust ventilation The condition is not caused by spongy or metallic platinum even in concentrations as high as 1,000 milligrams per cubic metre, nor is it caused by the complex salts of the allied metals palladium, rhodium and ruthenium

ORGANIC VEGETABLE MATERIALS

There are about 800,000 persons employed in agriculture in Great Britain, and it is therefore one of the largest industries in the country "Farmers' lung' was first described by Campbell (1932) and Fawcett (1938) in the north west areas, and by Fuller (1947) in Devonshire It has principally occurred in hay makers and has been called "bronchitis fenisecıorum" but it has been described in all harvest workers In Scandinavia, Tornell (1946) and Zettergren (1950) called it "threshers' lung" Hoffman (1946) reported the disease in the Canton of Appenzell in Switzer land during the harvest of 1945 There have been instances recorded where the infection has occurred in tea tasters A similar disease occurs in horses and is known as "broken wind" Fawcett ascribes the disease to fungi but though he clearly establishes the disease as an entity, the mycotic aetiology is not by any means proved There is no doubt that rotting vegetable material contains innumerable fungi, and if these organisms are in the dust which is inhaled they will also appear in the sputum They are constantly present in all the dusts which give rise to these conditions, and though they may not be the immediate cause it is possible that by producing toxic substances they indirectly contribute to the aetiology of the condition

Patients suffer from gradually increasing shortness of breath, which may get progressively worse over a period of months They may develop an acute febrile respiratory illness with a cough, scanty frothy sputum, and occasional haemoptysis Physical signs include patches of dullness and crepitations The blood sedimentation rate is raised Radiographic appearances in the earlier stages consist of a fine reticulation which is followed by a rather soft snowflake mottling, widely distributed throughout both lungs

When the disease progresses to the chronic stage the patient will gradually become more short of breath he will become cyanosed, develop clubbing of his fingers, and a cough with purulent sputum X ray examination at this stage will show an increase in the opacity of the mottling and an increase of the hilar and perihilar shadows coalescent areas of fibrosis, marked restriction of the diaphragmatic movements and considerable areas of emphysema

The patient eventually becomes emaciated and finally dies with right sided heart failure If diagnosis is made early the condition responds to treatment with potassium iodide, but if it is undiagnosed it progresses and the patient becomes a chronic invalid with fibroid lungs Necropsy shows voluminous lungs with emphysema and thickened pleurae with many bands of fibrous tissue traversing the lung substance

BAGASSE

Bagasse is sugar cane after the sugar has been extracted It is used for making boarding for interior decoration and thermal insulation One gramme of air borne

dust may contain up to 240 fungal spores Amorphous silica forms roughly 1-2 per cent of the bagasse but quartz only 0 1 per cent with many of the quartz particles being 20-30μ in size It is therefore impossible that there should be any risk of silicosis An account of the disease was first published by Castleden and Hamilton Paterson (1943) in England, and by Sodeman and Pullen (1943) in the United States, and it has been more fully described by Hunter and Perry (1946)

Workers exposed to bagasse dust for a period of 6 weeks or more develop an acute febrile illness with extreme shortness of breath, cough with scanty black stringy sputum, and occasional haemoptysis Throughout the lungs there is dullness, diminished air entry, and many course crepitations Skiagrams show miliary shadowing, which suggests an acute bronchiolitis (Fig 106) The acute illness carries a mortality rate of 10 per cent but the majority of patients make a complete recovery

In some, however, it progresses to a chronic stage in which the patient becomes excessively short of breath and develops a cough with much sticky sputum On examination these patients are orthopnoeic, cyanosed, and have clubbing of their fingers They have all the physical signs of emphysema with many coarse crepi-
[illegible] on shows many areas
[illegible] Necropsy, however,
[illegible] marked sub pleural
thickening

During the acute phase of the disease the patients are best treated with the usual methods used in the management of pneumonia, including sulphonamides and penicillin, and usually there is a marked response The treatment of the chronic condition is symptomatic and follows the principles of treatment of chronic bronchitis and emphysema

COTTON (BYSSINOSIS)

Jackson as early as 1818 drew attention to the fact that those who worked in the cotton industry, particularly card room workers, suffered from a characteristic respiratory disease, it also occurs in those who work with flax In 1862 Greenhow described the condition in a report to the Privy Council, and in 1932 the Home Office issued the Report of the Departmental Committee on Dust in Card Rooms in the Cotton Industry This report collected the existing information about the conditions in the mills, types of machinery in use, and the nature of cotton dust It included an account of the clinical examination and radiological findings in a group of workers at Ashton under-Lyne who suffered from the disease, and described the necropsy findings in a few cases The disease was named byssinosis from the Greek word βισσοζ meaning "fine linen" Foreign literature on the condition is scanty, but Zipperlen (1934) investigated patients suffering from asthma in the textile industry of Wurtemberg, and recorded that there were many with severe attacks in whom strong allergic reactions could be obtained [illegible]

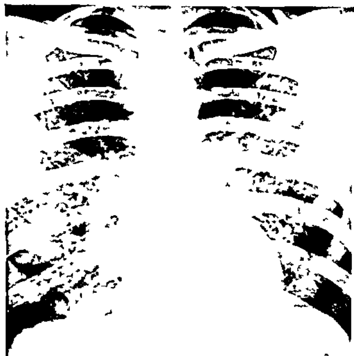


FIG 106 — Skiagram showing acute bronchiolitis caused by bagasse

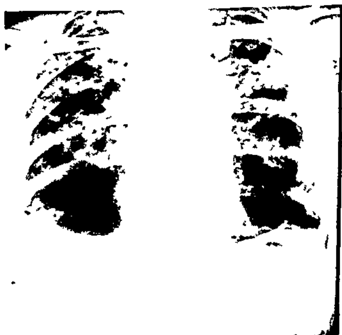


FIG 107 — Fibrosis of lung and bronchiol ectasis from bagasse

cotton industry He paid particular attention to the pathogenesis of the disorder but this is still a matter for debate The protein fraction of card room dust has been shown to be capable of producing inflammatory lesions in animals and normal men The characteristic course of the disease is suggestive of an acquired allergic hypersensitivity, and there is some confirmation of this view in the finding of positive cutaneous reactions to the proteins in affected persons, but only rarely in unaffected persons, but the primarily toxic qualities of the protein prevent the condition from being regarded as a purely allergic one Other factors that have been suggested as possibly concerned are a substance resembling histamine, and moulds Histamine-like substances similar to those found in card room dust have however been found in other dusts not causing similar symptoms Gill (1947) described the process of carding and the symptomatology of the disease

Cotton is obtained chiefly from Egypt, the United States of America, and India, and it arrives at the mill in tightly compressed bales, which also contain much foreign material such as particles of leaf and seed coat, cotton hairs, fragments of mould, and fine sand The raw cotton is therefore first cleaned, and the tightly compressed cotton is pulled off in layers and hand fed into the machine called the "hopper bale opener" It then passes into the blowing room where a series of machines, by means of beating actions and currents of compressed air, free it from most of the impurities All these machines are enclosed in dust-proof covers, the machines, however, have to be cleaned, and this is a particularly dusty job for which the workers have to be suitably clothed and wear respirators The clean cotton coming out of the blowing-room passes to the carding engines, and it is principally the strippers and grinders in charge of these engines who suffer from the disease Usually each stripper and grinder is in charge of 16 carding engines, each engine is cleaned four times daily by a vacuum process, and twice weekly it is brush stripped The latter process is necessary since the vacuum cleaning fails to clean sufficiently deeply The evidence is strong that the dust liberated by the carding process in the immediate vicinity of the carding engine contains the injurious dust which causes byssinosis The atmosphere is progressively safer the greater the distance from the carding machines

The usual history is that after working for several years in the dusty atmosphere the man begins to sneeze and develops a dry and irritating cough with a tight feeling in the chest and restricted intake of breath At first the trouble is temporary, passing off in 1–2 days, but it returns after a short absence from work such as a week-end It has, in consequence, been called "Monday morning fever", "mill fever" and "factory fever" Disablement and incapacity for work does not result at this stage, and the breathing will become normal on removal from the dusty atmosphere After exposure to the dust for 10 or more years, however, the symptoms become more severe and persistent, and the worker suffers from asthma and bronchitis associated with shallow breathing, cough, and a small amount of sticky, mucoid phlegm He loses weight and develops a sallow complexion Absence from work and incapacity increase, although removal from the dust causes improvement, with capacity to work in another atmosphere In the last stage the emphysema is severe Cough is present, with mucoid or muco-

purulent expectoration and great shortness of breath In extreme cases the man may stand with his hands pressed against his thighs to aid his breathing The disease is incurable at this stage and many of these workers die before the age of 50

The physical signs are those of chronic bronchitis and emphysema, the respira tion is laboured, with accessory muscles called more and more into use The thorax becomes typically barrel shaped, and the patient tends to sit in a charac teristic attitude with the body bent slightly forward the legs widely spaced, and the hands placed on the knees The vital capacity is greatly reduced and the

suggest considerable fibrosis (Fig 108)

Necropsies have revealed chronic bronchitis and emphysema but do not show any specific feature which enables the bronchitis of card room operatives to be distinguished from that which occurs in the general population Dunn and Sheehan (1932) have reported that dilatation of the right heart was frequent

No special medicinal treatment is of any value, and timely removal from the work is the only successful method of preventing the disease from progressing Respirators have been tried but are not satisfactory since the men find them difficult to work in and therefore do not wear them Methods of suppression of dust by exhaust ventilation and enclosing the machinery have been highly developed in the industry and the disease is thus kept to a minimum It is still however one of the most important industrial diseases at the present time

First Collis (1914) and then Middleton (1926) recorded that cotton weavers in Lancashire suffered from an acute bronchitis from handling cotton fibre, which was termed ' weavers' cough ' This was an acute illness with fever, cough and sputum, and residual symptoms, which might last up to two months The most probable cause of the outbreaks was thought to be the amount of moisture left in the warp, producing an abundant growth of fungi which developed during storage on the beams and produced, on weaving a dust laden with conidia spores and fragments of mycelium which were breathed by the weavers

In the United States of America Neal, Schneiter and Caminita (1942) described an acute febrile illness with cough, dyspnoea, tightness of the chest and more general symptoms occurring among workers making mattresses from low grade [illegible] plants where workers were [illegible] They thought the condition [illegible] *Aerobacter cloacae*, or its [illegible] This may be so but cotton [illegible] possibly be

CARCINOMA OF THE LUNG

Agricola and Paracelsus in the sixteenth century described at length a disease of the lungs with a high mortality rate in the miners of the Erzgebirge in Saxony The condition was known as " Bergkrankheit ' (mountain disease) It was not until



Fig 108 — Severe byssinosis in a man aged 50 years

1879 that it was identified by Härting and Hesse as cancer of the lung These authors attributed 75 per cent of all deaths of miners to the disease, which usually developed after a period of 20 years' work in the mines Arnstein (1913) distinguished the tumours as carcinoma, and differentiated them from lympho sarcoma and endothelioma, as most neoplasms of lung at this time were thought to be Since people in the district other than miners were only rarely affected by this disease, all the early workers attributed the condition to the occupation of mining The main ores in Schneeberg are sulphides and arsenides of nickel and cobalt — "Speiskobalt" ($CoAs_2$) "Wassnickelkies" (Ni_2As_2) and 'Rotnickelkies" (NiAs) — while at Joachimstal on the southern side of the Erzgebirge there is a high content of pitchblende and radium Rostoski, Saupe, and Schmorl (1926) showed that the bore dust of these mines contained up to 0 45 per cent of arsenic, while the dust in the pits contained 0 1 per cent , but it has not been found possible to prove that arsenic is either the cause or a contributory agent In Southern Sweden, where Speiskobalt is also mined pulmonary cancer is said to be unknown, and in Schneeberg no other signs of arsenic poisoning are reported Hueck (1939) has pointed out that multiplicity of tumours which is so frequent in arsenical cancer does not occur in Schneeberg miners, while estimations of arsenic in the urine, hair, and nails (Rostoski, Saupe and Schmorl, 1926), and in the lungs (Pirchan and Sikl, 1932 , Zeil, 1935), give equivocal results Work in the Joachimstal area by Loewy (1929) and Sikl (1930) has suggested that the radioactivity of the ores is the principal aetiological agent Peller (1939) showed, from



FIG 109—Man, aged 52 years nickel worker for 23 years Squamous cell carc noma of r ght upper lobe

necropsies that 50 per cent of the miners in this area died with lung carc noma while Lange *(1935) gave similar figures for Schneeberg Recently Lorenz (1944)* expressed the opinion that the radio activity could not be the sole cause of the carcinoma since similar doses of x rays or γ rays do not produce lung tumours in animals He suggested that both arsenic and radio activity m ght be contri butory *factors*

Amor (1938) reported 10 cases of carcinoma of the lung (Fig 109) and numerous cases of carcinoma of the nose and nasopharynx among workers exposed to arsenical dust for an average period of 6 years at a nickel works in South Wales *The ores were from Sudbury in Canada and contained 2 per cent of arsen c* and for a period of 25 years had had the copper extracted by the add tion of sulphuric [illegible] and calcina

pneumonectomy for carcinoma of the lung on 2 of these workers both were squamous cell carcinomas The arsenic contents of the lungs were 3 82 and 3 40 parts per million (p p m) the content of the hair of the men was 2 2 and 10 5 p p m *and they were excreting 41 5 and 39 8 micrograms per 24 hours*

Other evidence suggesting that arsenic produces carcinoma of the lung is the experimental work of Leitch (1922) who has succeeded in producing such growths in mice with potassium arsenite But the most conclusive evidence is the statistical work of Bradford Hill and others *(1948) which showed that in one town in England*

where the principal industrial work is the manufacture of arsenical sheep-dip the mortality rate for carcinoma in the chemical workers handling arsenic is double that in the rest of the population of the town, a statistically significant difference It was also shown that the difference is made up almost completely by carcinomas of the skin and bronchi

It is possible that the high content of arsenic dust in cigarette fumes may account for the world-wide increase in the mortality rate for carcinoma of the lung in males, which has risen six-fold in Great Britain in the past 10 years

Machle and Gregorius (1948) studied the mortality data for 7 chromate plants in the United States of America employing 1,445 workers They found 42 deaths from cancer of the respiratory system among a total of 193 deaths over periods varying with the different plants Of the 193 deaths, 66 were from cancer, thus the respiratory cancers were 63 6 per cent of all cancers, compared with 8 7 per cent in a control group of 733 deaths among Metropolitan Life Insurance Company industrial policy holders in the year 1946 Of these, 115 were from cancer, of which 10 were of the respiratory system The mortality rate for lung cancers in the chromate workers over 50 years of age was up to forty times that for a comparable industrial group In one plant exposure was restricted to bichromates and chromic acid, and here there was no cancer of the respiratory system, this evidence strongly suggested that the carcinogenic substances were the monochromates This also means that nasal irritation and septal perforation may occur without associated high rates for cancer of the lung The mean duration of exposure was 14 5 years The disease is further reviewed by Baetjer (1950)

Wyers (1950) has shown that there is an excessive mortality from carcinoma of the lung in cases of asbestosis In his series of 115 deaths from asbestosis 11

the more striking because 41 2 per cent of the deaths in his series were females, whereas the incidence of primary cancer of the lung in the general population is 4 4 males to 1 female

Workers with pitch and coal tar derivatives may also develop squamous cell carcinoma of the bronchus as well as epitheliomas of the skin

SILICATES

Most silicates will produce dust, and if workers are exposed to these dusts they

China-clay or kaolin

This is hydrated silicate of aluminium, and at present is one of the most important exports from the United Kingdom It is an important ingredient in china and earthenware pottery, and is used in the manufacture of a large number of other commodities, including cotton cloth, paint, soap, and fine art paper It

is formed by the decomposition of feldspar by the removal from it of silicate of potassium through the action of water containing carbon dioxide

The largest production of china clay in the world is in Cornwall, where it occurs in deep pits from which it is washed with water under pressure Men are exposed to the dust in shovelling the clay after it has been dried in the kilns No medical survey has been carried out in the Cornish china clay industry, and therefore little is known of the effect of this dust on the lungs Middleton (1936), however, recorded the case of a worker aged 49 years whose skiagram showed the whole of both lung fields covered with extremely fine mottling, most marked in the middle zones and at the right base He considered the film suggested pneumoconiosis, not of a silicotic type Hale (1946) has some radiographs of workers in the industry showing well-marked reticulation It is probable that these workers have few symptoms and that the dust does not produce great, if any, disability

Thomas (1952) described in the findings in two necropsies one a worker in the ball clay industry and one who had been engaged on grinding china stone, a granite containing china clay He found in these workers evidence of silicosis

Talc or French chalk

This is hydrated magnesium silicate, and in mineral form is known as steatite or soapstone It is used as a filler for paper, soaps, and paints, for leather dressing, in the manufacture of rubber motor tyres, in making electric switchboards, and for heat insulation of steam pipes

The first account of changes in the lungs due to talc was given by Thorel (1896) followed by a report by Cesa-biachi and Devoto (1911) of the necropsy of a woman who had worked in talc The lung showed much talc dust but little resulting fibrosis Zanelli (1931) recorded the case of a woman worker, aged 27 years, in a tyre factory, who had early tuberculosis but also reticulation in the lung radiograph Dreessen (1933) investigated the workers in a tremolite talc mine and mill and found that those who had been employed for more than 5 years had radiographs which showed fine diffuse bilateral fibrosis but this appeared to cause little disability Dreessen and Dalla Valle (1935) carried out a further survey in Georgia and found that 16 out of 33 mill workers exposed to high concentrations of talc and 6 of 13 miners had pneumoconiosis, in 4 cases there was evidence of added pulmonary infection Merewether (1933, 1934) examined 24 workers who were exposed to talc in the manufacture of rubber tyres for periods of between 4½ months and 40 years They showed no disablement though radiographic changes suggested diffuse interstitial fibrosis of the lungs (Fig 110)
Siegal, Smith and Gr [illegible] talc millers and miners
in St Lawrence Cou [illegible]
known as asbestine [illegible]
free silica content of [illegible]
contain fine, straight, needle like fibres, and dust counts ranged from 6 million to 5,000 million particles per cubic foot This material was obtained by underground mining with inclined shafts to depths as low as 1 000 feet, each mine employed 15–17 men underground, 221 miners and millers were examined and radiographic evidence of fibrosis was found in 32, giving an incidence of 14 5 per



Fig 110 — Woman aged 48 years exposed for 20 years to talc in a rubber tyre factory She complained of severe cough and dyspnoea

cent All the cases occurred in men who had worked for 10 years or longer, and the incidence in this group was 25 9 per cent The appearances were of fibrosis of a fine, diffuse type, showing granulation or nodulation on a hazy background The resulting dyspnoea, cough and fatigue were disabling, and many of the men showed clubbing of the fingers There was some increased susceptibility to tuberculosis 3 men showing evidence of the disease Besides the fibrosis, deposits of unidentified opaque material in the periphery of the lung called ' talc plaques " were observed in 6 3 per cent of the workers These plaques varied from single linear deposits in the region of the diaphragm to massive deposits, bizarre in shape, extending over a large part of the lung fields , they were irregular in shape, density, and the sharpness of their outlines No deaths were recorded, but the clinical and radiographic picture showed resemblance to asbestosis rather than silicosis McLaughlin, Rogers and Dunham (1949) recorded the results obtained at necropsy of a man aged 51 who had worked for 37 years in a rubber tyre factory, where he was exposed to talc dust A feature of the histology was the presence of bodies similar to asbestos bodies found in a pneumoconiotic lung Although there was much fibrosis of the lung and abundant doubly refractile particles, x ray analysis of the lung suggested that there was less than 0 06 per cent of free silica in the lung It seems clear that talc and asbestos give rise to similar diseases

Asbestos

The term ' asbestos " is of Greek *origin* and *means " unconsumable "* Asbestos

was worked into textiles by the Egyptians, Romans, and the inhabitants of the ancient empire of Tartary, the wealthiest among them using it as cremation cloth Pliny called it a "rare and costly linen", whilst Plutarch recalls its use as the perpetual wick in the lamps of the Vestal Virgins The use of asbestos seems to have been lost in the Middle Ages and an [illegible]
century met with little success, so
century that it again became indu[illegible]
is concerned with two main types of asbestos (1) Hornblende or amphibole, to which group blue crocidolite, readily identified by its lavender-blue colour, and amosite, white or yellowish brown in colour, belong, is produced exclusively in South Africa (2) Chrysotile, white in colour and of fine texture, is produced mainly in Canada, Rhodesia, Transvaal, Australia, Italy and Russia

TABLE II

TABLE OF COMPOSITION

	Hornblende		Chrysotile	
	Blue crocidolite, percentage	Amosite, percentage	Canadian, percentage	Rhodesian percentage
Silica	50	47	41	41
Alumina	1	6	3	1
Ferric oxide	35	37	2	4
Magnesia	2	6	40	39
Alkalines	8	1	—	—
Water	4	3	14	15

It is imported as the crushed rock, but the fibres can be separated to an extreme degree of fineness which renders them capable of being carded, spun into yarn and woven into cloth by processes similar to those used in the cotton industry Besides being woven, asbestos is ground and mixed with cement and plastics to make insulating slabs, boarding, and many other articles used in industry The dust given off during the process of manufacture is mostly less than 5μ in diameter and 10μ in length

The first case of asbestosis was described by Murray (1907) The man had been employed on a carding machine for 14 years and was the last survivor of 10 men who had worked in the card room, the others all having died about the age of 30 with respiratory diseases He died in Charing Cross Hospital in 1900 at the age of 34 Necropsy revealed extensive diffuse pulmonary fibrosis and the lungs were found to contain asbestos bodies In 1906, Marchand and Reisal (Kruger, Rosloski and Saupe, 1931) noted the presence of unusual bodies in the lungs of an asbestos worker, while Auribault (1906) in France recorded the high mortality in an asbestos textile factory [illegible]
in Hamburg Real attention, however,
who recorded the death of an asbesto
and pulmonary tuberculosis, while McDonald (1927) and Cooke and Hill (1927)

gave the first detailed descriptions of asbestos bodies and asserted that they originated in the inhaled dust Seiler (1928), and with Gilman in 1931, recorded the necropsy findings in an asbestos worker who showed fibrosis but no evidence of tubercle The condition was fully described by Merewether and Price (1930) and Merewether (1933, 1934) Clinically, the striking symptom is dyspnoea of gradually increasing intensity, often associated with a dry cough, in the early stages physical signs are slight or absent, but later râles throughout the lung develop and the fingers often become clubbed Radiologically the most characteristic feature is the "shaggy" outline of the heart shadow, while in the which
which

At necropsy, pleural adhesions are extensive and dense, and there are often thick subpleural plaques of fibrous tissue The consistency of the whole lung is increased, and there may be large areas of fibrous condensation which is tough rather than stony hard as in silicosis Other parts of the lung, not affected by the fibrotic change, are congested and consolidated with inflammatory processes (Figs 112 *a* and *b*) Emphysema is extensive, but localized often to the lower and apical parts of the lung, pneumothorax sometimes occurs The histology has

Wood and Gloyne (1934) collected 100 cases of the disease and found that 21 were complicated by pulmonary tuberculosis, but Middleton (1936) studied 54 deaths occurring between 1930 and 1934 and found that tuberculosis was present in 24 (44 per cent) The periods of employment in the industry ranged from 10 months in the disintegrating plant to 23 years' weaving, the average duration being just under 10 years He suggested that tuberculosis was a slightly less frequent complication than in silicosis and that it is more frequent as a terminal complication than as a concomitant disease during the earlier stages

Wyers (1947) showed that, as a result of measures enacted by the legislation of 1931, the morbidity rate from asbestosis fell from 39 1 per 1,000 in 1932 to zero

the disease
cancers)
and Wedle
characteristic of this disease, has now largely given place to a coarser, more granular appearance Pathologically, confluence of the blue polygonal areas is not so



FIG 111—Man aged 56 years employed for 6 years carding in an asbestos factory *He had gross clubbing of fingers and dyspnoea*

advanced as that seen in the specimens of 15 years ago Wyers thought these changes were due to longer exposures to lower concentrations of dust

From his clinical studies of asbestosis Wyers concluded that it differed from silicosis in being a clinical rather than a radiological disease Of the clinical signs he stressed the importance of clubbing of the fingers and symptoms of *cardiovascular* origin (*raised diastolic blood* pressure and *accentuated second* pulmonary sound) as being always present when once established whereas the marginal crepitations and fibrotic crackles tend to be evanescent Of the complications emphysema was always present bronchopneumonia the commonest termination tuberculosis was of the acute caseous type and occurred in one third of the cases the right heart dilatation was common and bronchiectasis rare Pregnancy could not be shown to affect the progress of the disease adversely and the obstetrician should be guided by cardiovascular rather than the pulmonary signs

The difference between the reaction of the tissue to silica and to asbestos is even more marked than the difference between the clinical pictures of silicosis and [illegible]

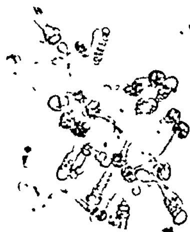
dust does not cause an acute inflammatory reaction Gardner and Cummings (1931) found that asbestos accumulated in the bronchioles and then in the alveoli and fibrosis first appeared after about 500 days exposure Gardner (1942) put



(a)



(b)



(c)

FIG 112—(a) Lung of an asbestos spinner (a patient of Dr H Wyers) (b) lung of asbestos worker showing gross thickening of pleura (a patient of Dr H Wyers) (c) asbestos bodies in sputum

forward the 'mechanical' theory as an explanation of the disease It was the long fibre in the dust which caused asbestosis, and if the asbestos were ground sufficiently finely no disease resulted He concluded that the disease results from the mechanical irritation produced by a combination of the long fibre and the rhythmic movement of the lung He also showed that different pathogenic responses resulted in different animals King, Clegg and Rae (1946), however, produced somewhat different results from the intratracheal injection of asbestos dust into rabbits they reported that the injection of long fibres (15μ) produced a nodular reticulinosis comparable with the experimental silicotic nodule, whereas short fibres (2 5μ) produced a diffuse interstitial reticulinosis

Silimanite

Silimanite is an aluminium silicate, and is a dense rock found in India It is used as a refractory material in the manufacture of porcelain When broken it produces masses of prismatic crystals in the form of fibres which cannot be broken down into fine fibres like asbestos but tend to break and become reduced to a powder

When handled in industry the rock is crushed, ground and sieved, and then calcined, during which a considerable quantity of dust is given off Middleton (1936) reported the investigation of 15 men who were employed handling this material Their ages ranged from 24 to 50 years and the duration of their employment in the industry was from 1½ to 16 years Chest radiographs showed abnormality in only 4 men, who had been working with the material 16, 11, 7 and 5 years, and in whom some reticulation was noted These numbers are small, but [illegible]

5μ in diameter Necropsies were carried out on 22 of these animals Five died from collapse or pneumonia after being dusted for periods of up to 100 days, and showed minimal fibrosis, 14 survived for periods up to a year, and in this group there was more fibrosis with thickening of the inter-lobular septa Three of the animals survived 673 days, these showed well defined dust nodules in the inter-lobular septa and in the perivascular and peribronchial regions, consisting of dust particles with fibroblast and early connective tissue formation These animal experiments suggest, therefore, that the dust may not be quite as harmless as the clinical observations of Middleton suggest

Mica

Mica belongs to a group of widely distributed rock-forming minerals, of which muscovite and phlogopite are important commercially Muscovite is a silicate of aluminium with potassium and phlogopite is magnesium-aluminium silicate The former is used for its transparency and the resistance to fire for windows of stoves and lanterns while it is sometimes ground up and used in the manufacture of paper, wall-paper, and paint, and as a lubricant and absorbent The latter is used exclusively for electrical purposes Dust is produced when the materials are being ground and finished

Middleton (1936) reports that Ferguson examined 12 workers who were exposed to mica dust, but only 5 of them had been exposed for more than 5 years and these complained of cough and shortness of breath Radiographs showed some fibrosis of the peribronchial type with some diffuse shadows in the middle zone Ferguson thought therefore, that the dust of mica was probably capable of causing pulmonary fibrosis

Diatomaceous earth and tripoli

Diatomaceous earth and tripoli are hydrated, amorphous minerals of organic origin, which are generally considered to be varieties of opal Diatomaceous earth is also known as diatomite, kieselguhr and ferina fossil It is found in the lake deposits of mineral diatomaceous shells It forms a porous mass sometimes

loose and floury, sometimes compact and clay like, and is always light-coloured, either white, grey or yellow. Tripoli, on the other hand, is a marine sediment. It generally contains well preserved fossils of fish and shell fish. After quarrying, draining and pulverizing, diatomaceous earth is marketed for a variety of industrial purposes including the manufacture of refractory bricks, and filters for drinking water, wines or syrups. It is used as an insulating material. Tripoli has fewer uses, but it is used for polishing metals and precious stones. Fuller's earth is also a mineral substance containing silica. Legge and Rosenkranz (1932) recorded the

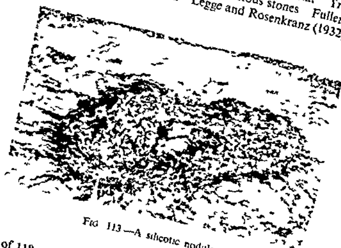


FIG 113.—A silicotic nodule

investigation of 118 workers in a diatomite quarry in Santa Barbara, California; 81 of the patients showed radiological abnormality and in these the changes were advanced. The patients showed clubbing of the fingers and complained of shortness of breath. More recently Martin, Valland and Champeix, in 1947, examined 32 workers exposed for many years to heavy concentrations of diatomite dust, but only 3 showed x ray appearances of mild interstitial pneumoconiosis, and of these only one had any disability. They concluded that while diatomaceous earth produces radiological and clinical disease it was of a comparatively mild variety. Vigliani and Mottura (1948) recorded 8 cases in which a severe pneumoconiosis developed after as little as 5 years exposure to the dust. A peculiarity of these cases was the tendency to spontaneous pneumothorax. Cases of pneumoconiosis resulting from long exposure to Fuller's earth have been recorded by Middleton (1938) in which Gloyne showed that in the lower part of the lung there were extensive patches of palpable dust fibrosis.

SILICOSIS

Silicosis is a fibrosis producing lung disease caused by the inhalation of free, crystalline silica or quartz particles. It is the most disabling of all the dust diseases. It was originally thought that the disease was caused by the mechanical action

of the sharp particles of quartz, but Gardner (1932) showed, by animal exp ments, that silicon carbide (carborundum) the particles of which are as sharp those of quartz, does not produce fibrosis to the same degree These experime resulted in the solubility theory of silicosis Quartz is taken up by the phagocy

TABLE III

FATAL CASES INVESTIGATED UP TO THE END OF 1949

(From the Annual Report of Chief Inspector of Factories 1949)

	Number of deaths	*Average age at death*	*Duration of employment in years*		
			Longest	*Shortest*	*Averag*
SILICOSIS					
Pottery					
Silicosis	579	61 2	62 0	2 8	38 8
Silicosis with tuberculosis	423	55 5	67 0	5 0	34 6
Sandstone					
Silicosis	206	59 2	60 0	9 0	39 4
Silicosis with tuberculosis	209	56 7	55 0	5 0	37 2
Grinding of metals					
Silicosis	102	58 7	56 0	14 0	36 4
Silicosis with tuberculosis	196	53 7	53 0	2 8	32 9
Sandblasting					
Silicosis	61	48 7	28 0	1 7	12 3
Silicosis with tuberculosis	96	46 1	46 0	2 0	13 1
Manufacture of scouring powders					
Silicosis	13	37 2	37 0	2 3	8 2
Silicosis with tuberculosis	6	40 8	11 2	2 0	7 0
Miscellaneous					
Silicosis	211	54 6	57 0	1 5	23 3
Silicosis with tuberculosis	206	51 2	50 0	0 7	25 2
Total					
Silicosis	1 172	58 5	62 0	1 5	34 2
Silicosis with tuberculosis	1 136	53 8	67 0	0 7	31 1
ASBESTOS					
Asbestosis	186	48 2	48 0	0 5	15 6
Asbestosis with tuberculosis	78	39 2	29 0	0 8	10 5

in which it goes into solution So long as it is contained within the phagocytes it is harmless but when it is released as a result of infection, either tuberculous or non-tuberculous, the typical whorled fibrous nodule of silicosis results (Fig 113) This stimulated the work of Kettle (1932) who showed that by mixing iron oxide with silica the amount of the fibrosis that was produced in the lung was reduced Later Denny, Robson and Irwin, in 1938, showed that aluminium-coated quartz particles did not go into solution This led them to use powdered aluminium for the prevention and treatment of silicosis, fine black metallic aluminium powder with particles of less than 2μ being blown into the workers changing rooms before they descend the mine The solubility theory is, however, far from satisfactory since silicic acid normally appears as an inert substance It seems probable there

fore that the disease is brought about by some physico-chemical process at present not fully understood (Hefferman, 1948) It is clear, however, from these observations that the effect of silica on the lungs is greatly modified by mixture with other dusts In industry silica rarely occurs in a pure form, and the type of disease that results from exposure to it is greatly modified by the substances with which it is mixed, as is well shown by Gloyne (1951) in his review of the pathology of 1,036 pneumoconiotic lungs Pure silicosis occurs in workers engaged in metalliferous mining granite quarrying, metal grinding where sand stone is used, and among sand stone masons The most rapidly developing variety of silicosis that has been described was that encountered in workers engaged in the manufacture of abrasive soaps and scouring powders, where it is possible that the presence of an alkali accelerates the action of the silica (Macdonald, Piggott and Gilder, 1930) Silicosis also occurs in the workers in the underground slate quarries in North Wales, but it has not been reported from the ancient quarry in Cornwall This condition has been called schistosis



Fig 114 —Macroscopic appearances c Fig 121 African goldminer after 2(years work showing typical whorled nodules and the conglomerate area at the apex (Section cut by Professor Jethro Gough)

Pathology

The classical pathological picture of silicosis is that of a discrete nodular fibrosis (Fig 114) The fibroblasts forming the nodule have a typical laminated appearance, which causes the structure of the nodule to resemble the cut surface of an onion The nodules vary in size from 0 5 centimetre to 1 0 centimetre in diameter, but they can be seen and palpated when they are only from 1 millimetre to 2 millimetres across These are characteristically of smooth black or marbled grey black colour and may be divided into three groups Minimal silicosis in which the whorled nodules can be detected only under the microscope, nodular silicosis in which the nodules stand out from the cut surface of the lung most numerous in the upper lobe and with a tendency to occupy the peripheral part of the lung, thus showing a pleural drift And lastly massive silicosis where, as the disease progresses, the nodules in some areas generally in the upper part of the lung, become more numerous and tend to conglomerate to form large hard masses The centre of the nodules tends to undergo a degenerative process and therefore to lose the characteristics seen in the early stage The surrounding lung tissue in classical uncomplicated silicosis shows little evidence of interstitial fibrosis The

t the root of the lung undergo a process of fibrosis They
h hard and are usually deeply pigmented and histologically show
sis in which the fibroblasts are arranged in a whorled manner
ndergoes a fibrotic change resulting in thickening and the formation
This may result from the deposition of silica in the subpleural
sult of a retrograde lymphatic flow

ust have been exposed to silica dust for a period of probably more
This may have occurred in some early part of his life, so that a
pational history must be obtained He complains of increasing
breath on exertion, and this may be exaggerated by a super added
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tightness of the chest and a wheeze Not only does tuberculosis pre-
licosis as has been recorded above but silicosis predisposes to the
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ve diminished costal and diaphragmatic movements There may be
the fingers, rhonchi will be heard scattered throughout the lungs
expansion may be reduced to less than an inch The blood sedimentation
will be increased if infection is present and it would appear that infection and
disability are closely allied The vital capacity and maximum breathing
capacity are reduced, the residual air increased, the blood oxygen tension is
reduced Skiagrams of the chest will show reticulation, nodulation and in many
case massive shadows, which are caused by the conglomeration of the whorled
of fibrosis Radiologically, similar shadows may arise in diseases such as
miliary tuberculosis, sarcoidosis or haemosiderosis It is not possible to dif
ferentiate these various diseases by examination of the skiagram alone A
complete clinical and occupational history is essential for the diagnosis of silicosis
(Figs 115–121)

Treatment

Once the disease is established treatment can only be symptomatic If the patient is freed from contact with silica before massive shadows have developed the disease will not progress, but if massive shadows are present it may continue to progress even though he is no longer in contact with silica dust Many workers hold the view that massive shadows are a manifestation of tuberculous infection even though it is usually impossible to isolate the tubercle bacillus Gloyne (1951) reported that in 1,036 pneumoconiotic lungs which he examined, he found evidence of pulmonary tuberculosis in 43 per cent He found primary cancer of the lung in 7 7 per cent Apart from removing the man from contact with the silica, treatment should be directed to treating infection There is no scientific evidence that treatment by inhalation of aluminium powder has any therapeutic effect in relieving symptoms or preventing the disease, though workers and patients usually express satisfaction with the results of the treatment

Prevention

Prevention is the most important form of therapy Managements should be



FIG 115.—Silicosis in an iron moulder Reticulation



FIG 116.—Silicosis in a steel fettler Nodulation

lymphatic glands at the root of the lung undergo a process of fibrosis They become small and hard and are usually deeply pigmented and histologically show a process of fibrosis in which the fibroblasts are arranged in a whorled manner The pleura also undergoes a fibrotic change resulting in thickening and the formation of adhesions This may result from the deposition of silica in the subpleural lymphatics as a result of a retrograde lymphatic flow

Clinical course

The patient must have been exposed to silica dust for a period of probably more than 5 years This may have occurred in some early part of his life, so that a complete occupational history must be obtained He complains of increasing shortness of breath on exertion, and this may be exaggerated by a superadded catarrhal bronchial infection, which may give rise to cough and sticky sputum and later to tightness of the chest and a wheeze Not only does tuberculosis predispose to silicosis as has be[illegible] to the development of tuberculosis [illegible]

On examination the chest [illegible] shaped and have diminished costal and diaphragmatic movements There may be clubbing of the fingers, rhonchi will be heard scattered throughout the lungs the chest expansion may be reduced to less than an inch The blood sedimentation rate will be increased if infection is present and it would appear that infection and disability are closely allied The vital capacity and maximum breathing capacity are reduced, the residual air increased, the blood-oxygen tension is reduced Skiagrams of the chest will show reticulation, nodulation and in many cases massive shadows, which are caused by the conglomeration of the whorled areas of fibrosis Radiologically, similar shadows may arise in diseases such as miliary tuberculosis, sarcoidosis or haemosiderosis It is not possible to differentiate these various diseases by examination of the skiagram alone A complete clinical and occupational history is essential for the diagnosis of silicosis (Figs 115–121)

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F G 115—S l cos s n an iron moulder Ret culat on



FIG 116—S l cosis n a s eel fettler Nodu lat on

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FIG 117.—Reticulation and massive shadows in a brass fettler



FIG 118.—Reticulation in a tin miner



Fig 119 — Reticulation and massive shadows in a granite quarry man aged 28 years



Fig 120 — Nodulation massive shadows and opacity of mediastinal lymph glands resembling a bunch of grapes in a tin miner

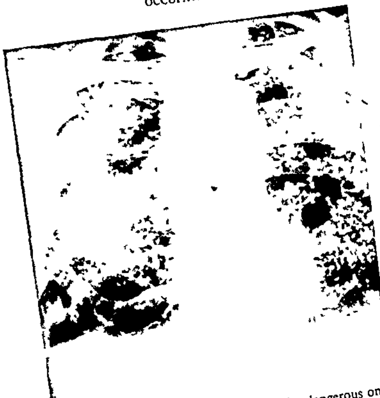


Fig 121 —Nodulation massive shadows, opacity of mediastinal lymph glands and basal emphysema in a South African gold miner (a patient of Dr L W Hale)

encouraged to substitute safe processes for dangerous ones, as for instance in the use of wet instead of dry methods of quarrying, blasting, drilling and other processes Dangerous substances used in industry should be replaced by safer substances Thus aluminium oxide should be used to make grinding wheels instead of sandstone Mechanical methods should be used to suppress dust This may be done by the use of wet processes, and exhaust ventilation should be installed to remove any dust which has formed Use should be made of efficient respirators so that the dust is removed from the air which the man breathes Suitable provisions should be made so that the workers can have their meals away from the machinery and the dusty atmosphere There should always be adequate medical supervision If the engineer could prevent the creation of dust there would be no disease

GRAPHITE

No systematic survey of graphite workers has been carried out but Dunner (1945) described the clinical and radiological findings in men who had been exposed to pure graphite dust unloading ships for periods of about 20 years Three of the men had cough, sputum and shortness of breath, but two had no complaints X ray examinations showed reticulation and nodulation Later Dunner and Bagnall (1946) reported a case of “ necrosis cavitation ” in the lung of a graphite worker The sputum contained graphite although the patient had not worked for 5 years in contact with this substance During recent years the graphite used in Great

Britain has come mainly from Ceylon and Madagascar but there has been a small quantity mined in Borrowdale in Cumberland Dassanayake (1948) showed that the miners in the workings in Ceylon developed x-ray reticulation Gloyne, Marshall and Hoyle (1949) recorded two fatalities and Harding and Oliver (1949) three more among men who had been exposed during the manufacture of crucibles to a mixed dust of graphite, silica, sillimanite and other substances The patients had worked for 30 years and more in contact with the dust and had complained of increasing shortness of breath on exertion, with cough and viscid sputum Chest movement was poor, there was diminished air-entry, and many crepitations throughout the lungs The radiographs showed emphysema at the bases, nodulation, and massive shadows of both lungs The patients all had massive progressive fibrosis, and dust reticulation at necropsy, and the lungs were blackened from the presence of graphite dust

PNEUMOCONIOSIS OF COALMINERS

Pneumoconiosis of coalminers (anthracosis) is the most important dust disease in the British Isles The existence of the disease in South Wales has been known for a century (Meiklejohn, 1951) Early in the nineteenth century Laennec (1806) and Boyle (1815) described a peculiar form of animal production which they called melanosis Thackrah in 1831 recognized that colliers were liable to develop " miner's asthma " and Cox (1857) described the disease as the " scourge of the mines " , but it was not until Hart and Aslett (1942) carried out a survey of the industry, as a result of the rapidly increasing number of compensation certificates that were being issued, that a clear description of it was recorded in the literature The dust inhaled by the coalminers is a mixture of coal, silicates, and silica and the resulting disease is therefore a form of silicosis Besides the mixture of dusts the quantity of dust inhaled is a factor in the occurrence of disease In the past the anthracite mines have been badly ventilated and therefore the quantity of dust inhaled has been high and the incidence of the disease has accordingly been highest in these mines However, with the installation of better ventilation and the use of mechanical methods in bituminous and steam coal mines the quantity of dust in the atmosphere in all classes of mines has approximated more closely together, and there is probably not such a great difference in incidence as used to be thought

Pathology

If the lungs are examined, black areas will be found scattered throughout the lungs, varying from microscopic size to about 5 millimetres, some of which are soft, others are hard, depending on the amount of fibrous tissue present (Fig 122) These dust foci are formed around the bronchioles and their accompanying arterioles A delicate fibrosis develops in the foci of dust which may stop at this stage or proceed to the development of hard collagen tissue These foci have a crenated edge with processes extending from the edge and fading into unaffected



FIG 122—Macroscopic appearance of a section of lung of a miner with pneumoconiosis showing coal foci with areas of surrounding emphysema and massive apical area of fibrosis with cavity in the centre (Section by Professor Jethro Gough)

1947) It is the destruction of healthy lung and its replacement by fibrosis and focal emphysema which cause the shortness of breath and consequent disability in this disease

As a background to this simple pneumoconiosis there may be super imposed fibrosis due to infection which may be tuberculous or non tuberculous Massive areas of fibrosis occur frequently in the upper halves of the lungs especially in the posterior segment of the upper lobe and the apical segment of the lower They are round or elongated and are composed of dense black tissue of a hard rubbery consistency These areas usually have a small cavity in the centre, which may contain some black liquid There is usually no definite evidence of tuberculosis either to the naked eye or to microscopic examination Nevertheless it is thought by many workers, probably rightly that these areas do represent super added tuberculous infection which may have become dormant or died out This view is supported by the finding of tubercle bacilli in about one third of the cases Sometimes these areas extend into the bronchus, in which case the patient may complain of coughing up a quantity of inky black sputum This has been called melanoptysis

Right sided heart failure is the most common cause of death in these patients but they may die from tuberculo toxaemia or thrombosis of the pulmonary artery The degree of disability will depend on the amount of lung in which function has been lost or impaired This may be brought about either by fibrosis or

emphysema, although the two generally occur together The disease presents most rapidly in the workers who are employed in developing the roads to the coal face and are called "hard headers", but it can occur in all workers in the coal mines, or indeed, in coal trimmers working in the docks, or even in furnace men in factories, and coal-delivery men, if the exposure is long enough The disease is similar to that already described in the section dealing with graphite

Clinical course

In the early stages the disease tends to pursue a slow course of dyspnoea on exertion, and this may be the only symptom As the disease progresses, however, they may develop a cough, and advanced cases may have melanoptysis, and cough up a peculiar type of black sputum This is associated with the breaking down of large areas of diseased lung tissue In the late stages, paroxysms of coughing associated with tenacious mucoid sputum sometimes occur On examination in the early stages there may be no abnormal physical signs, but as the disease develops the patients become obviously dyspnoeic on exertion and eventually orthopnoeic There may be clubbing of the fingers The chest tends to become barrel-shaped, and respiratory movements are poor Flattening of the *chest below the clavicles is sometimes marked , there is often hyper-resonance* over the bases Air entry becomes markedly diminished, and when bronchitis is superimposed there will be coarse crepitations throughout the lungs

The progress of the disease is often accompanied by a rising blood sedimentation rate, but this is usually associated with infection It is, however, the superimposed infection, which may be either a catarrhal bronchitis or pulmonary tuberculosis, which causes the disability in the condition Tuberculosis is a common complication and Mann (1951) suggests that it is possible that coal dust exerts a modifying influence on its development and progression Stewart (1948) found that positive sputum was present in 3 per cent of 400 patients with the disease , but Gloyne (1951) found pulmonary tuberculosis in 100 (34 per cent) out of 293 coal miners' lungs examined at necropsy He found that the incidence of primary pulmonary neoplasm was 6 9 per cent The vital capacity and maximum ventilatory capacity are usually below normal and the residual air is increased Exercise tolerance tests are below standard Measurements of arterial oxygen will show lowered saturation and tension in advanced cases In the early stages the skiagrams show

near the hila In more advanced cases nodulation is present , multiple, discrete shadows 2–3 millimetres in diameter are scattered throughout the lung fields (Figs 124–125) This nodulation in due course may coalesce or conglomerate so that massive shadows develop These are dense homogeneous shadows usually with a smooth outline, and therefore well-defined, often with a symmetrical distribution, and sometimes referred to as "angel's wings" These shadows are usually associated with well marked basal emphysema They can occur either with reticulation or nodulation Following melanoptysis, a central area of translucency develops within a massive shadow, indicating the presence of a cavity There is sometimes a fluid level (Fig 126)

If the man is removed from the mines in the early stages of the disease it is not progressive In the later stages it frequently pursues a slow course, but when massive shadows are a prominent feature it may progress rapidly.

Treatment

When the diagnosis is made the patient should be removed from further exposure to coal dust If this is done in the early stages he will be able to do full time work in most industries As the disease advances he will be suitable for work in light industry or in sheltered workshops Treatment for the chronic bronchitis and emphysema which accompanies the disease follows the general line for treatment of these diseases Inhalation of aluminium dust may cause the patient to express the faith that he is better, but there is no scientific evidence that it produces any change in the course of the disease

PNEUMOCONIOSIS

It is only since 1930 that radiography of the lungs has been satisfactory It is not surprising, therefore, that much new knowledge about the effect of dust on the lungs has accumulated during the past 20 years The characteristic radiographic change attributed to pneumoconiosis is pin-point shadowing scattered throughout the lung In the description of this nearly every worker has found it ... a new one of

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either fluffy and confluent, or else conglomerate, when a massive shadow results ... of the x-ray ... a soft

that is seen on an x-ray film There may be no visible difference between ... nodulation caused by deposits of iron in the electric arc welder's chest, and that appearing in the classical silicotic or even in the person suffering from chronic miliary tuberculosis, yet the pathological lesions are fundamentally different The observer gives his own opinion of the cause of the shadowing

Even though many of the dusts which cause this shadowing are inert, it is rational at least to suppose that the lung cannot become a physiological dust trap and yet retain its elasticity It seems possible that any dust, no matter how ... in small concentrations would in large enough quantity eventually

Inert dusts

Baratosis

When metallic dust is inhaled into the lung it may go into solution and be carried around the body and excreted by the kidneys and intestines, or it may remain in the lung tissues as an inert substance *Metals and their salts throw x-ray shadows,* the densities of which vary with their atomic weight *Arrigoni* (1933) and Pender-

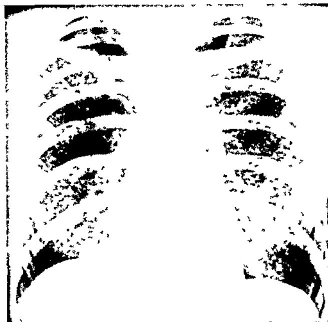


Fig 123 —Early reticulation in a South Wales collier

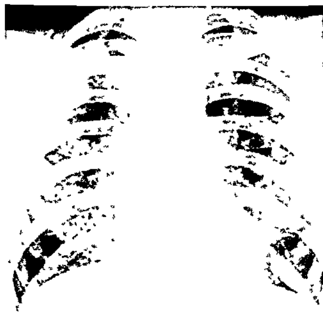


Fig 124 — More definite reticulation and early massive shadow formation in a South Wales steam collier

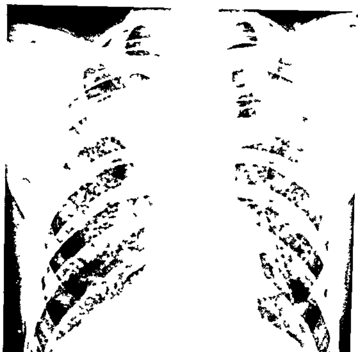


FIG 125 — Nodulation and massive shadow formation in South Wales anthracite hard header aged 48 years



FIG 126 — Skiagram showing a cavity which appeared in a massive shadow in a coalminer's lung after a melanoptysis

grass (1938) have shown that the inhalation of the dust of barytes and barium (atomic weight 137 4) produces the densest of nodular shadows in the lung. Barium itself, however, is inert and does not produce symptoms or disability. If, however, the dust is a mixture containing quartz, silicosis may result.

Siderosis

Zenker (1866) first showed that iron (atomic weight 55 84) was deposited in the lymphatics of the lung tissue. Doig and McLaughlin (1936) describe the radiographic change which they observed in a group of 6 electric-arc welders (Fig 127). These men suffered no symptoms and they had no physical signs as a result of inhaling the iron dust, yet their skiagrams showed dense reticulation. Enzer and Sander (1938) showed at necropsy that the iron oxide is deposited in the perivascular and peribronchial lymphatics and does not produce fibrosis in the lung. Later Doig and McLaughlin (1948) showed that the x ray shadowing will become less marked in the course of years if the men are taken away from the occupation of welding. The study of dust diseases is inevitably complicated by the fact that the dust inhaled is of a mixed nature. Thus workers in foundries are exposed to a mixed dust of iron, silicates and silica (McLaughlin and others, 1950). In these cases it is manifestly impossible to express an opinion as to how much of the x-ray shadowing is due to the inert iron dust, and how much to fibrosis caused by the silica and silicates (Fig 128). Likewise though the radiographic changes in the lungs of haematite iron miners are highly characteristic, these workers are exposed to a mixture of iron and quartz, resulting in sidero silicosis with all the symptoms and disability caused by the more severe disease (Fig 129).

Argyro siderosis

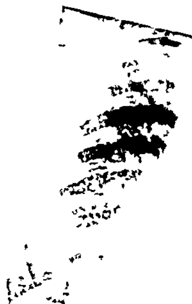
Silver polishers polish with rouge, and are thus exposed to a dust consisting of a mixture of silver (atomic weight 107 88) and iron. They develop a cough with copious rouge coloured sputum. McLaughlin and others (1945) showed radiographically that the lungs of these workers presented a typical picture of reticulation. No evidence of fibrotic change was shown though the lungs may be emphysematous. There were collections of inert iron in the lymphatics. Histologically there is intra-vitam staining of the elastic tissue in the arterial and alveolar walls from the inhalation of particles of silver, which is partly the cause of the x ray shadowing (Fig 130).

Stannosis

Pendergrass and Pryde (1948) reported the case of a workman, aged 45, who had been bagging tin (atomic weight 117 8) oxide dust obtained by treating tin cans with a caustic. The inhalation of the dust produced radiographic nodulation in the lung fields similar to that seen in arc welders. There was little fibrosis. The tin oxide dust contained 96 5 per cent of tin oxide but no silica. When an artery of a freshly excised dog's lung was injected with a saline suspension of tin oxide or the suspension was instilled into the bronchi even small amounts of it produced appreciable densities in the lung.

Vanadiosis

Wyers (1946) has shown that men employed in bagging vanadium pentoxide in




F o 127 —S deros s in an electr c arc welder aged 60 years af er 21 years of the work

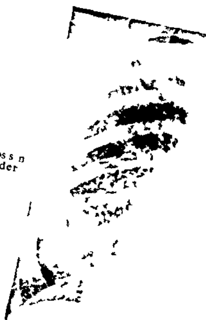



FIG 128 —S l cos s n steel moulder Nodulat on



Fig 129 —Appearances of s dero s l cos s in a haemat te iron ore m ner

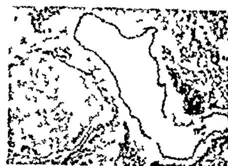
the process of manufacturing vanadium (atomic weight 50 6) may develop rad ographic reticulation in their lungs

Aluminium

Aluminium powder has been used extensively in making explosives incendiary mixtures and paints Denny Robson and Irwin (1939) produced evidence from animal experiments that aluminium dust would prevent silicosis Since then miners in various parts of the world have had aluminium dust blown into their changing

from Germany have shown that workers exposed to high concentrations of metallic aluminium dust complained of dry cough with pain on breathing shortness of breath and poor appetite Their skiagrams show a rapidly progressive disease with extensive fibrosis (Fig 131) These patients frequently developed spontaneous pneumothorax and several d ed The development of the illness was reported within 3 months of starting work Histolog cal examination of the lungs of patients who d ed revealed coarse branching hyaline collagenous fibres wh ch enclosed phagocytes containing fine and coarsely granular particles distinguished by jagged outline from carbon part cles

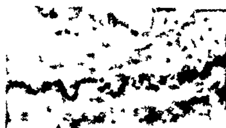
In 1947 Shaver and Riddell in Canada reported that a number of persons



(a)



(b)



(c)

Fi ^ r or

sect on before removal of the s lver (× 1 600 *reduced one quarter (approx) on reproduct on)*

engaged in the manufacture of aluminium oxide for abrasive wheels develop·d a similar disease In the manufacturing process bauxite is ground and mixed intimately with iron and coke the mixture is shovelled into large iron pots into which carbon electrodes are lowered The mixture is fused at about 2 000 C as a result of which dense white fumes contain ng alumin um and s lica are evolved These fumes contain considerable quantities of alumina and silica together with *small quantities of many other substances all in a fine state of division* The men first develop attacks of extreme shortness of breath and later compla n of sub sternal pain cough with white frothy sputum weakness loss of appetite and marked cyanosis Among 344 men 23 developed symptoms and 8 developed bilateral spontaneous pneumothorax There were 7 deaths in these cases the men had had less than 5 years exposure The chest s gns were variable and depended on the presence or absence of pneumothorax d minished chest expans on and impaired percussion note were the principal ones observed rales and rhonchi were variable Tachycardia and cyanosis were marked features In well established cases the rad ographs showed extensive fibrosis w th areas of gross emphysema The lung fields were lace like or granular in appearance Emphy sematous blebs and bulli occurred in the visceral pleura and were apt to rupture spontaneously giving rise to pneumothorax M croscopical exam nat on of the lungs revealed profound emphysema accompanied by overwhelming fibrous tissue formation The ash of the lung contained 3 per cent s lica and 45 per cent alumina

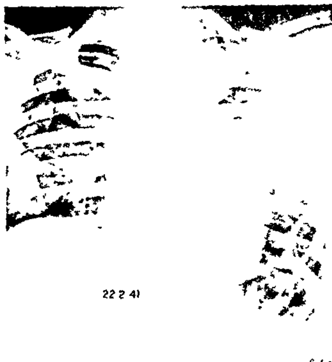


FIG 131 - Skiagram of man aged 38 years an aluminium stamper for 6 months showing extensive fibrosis of lung and spontaneous pneumothorax

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CHAPTER 24

CYSTS OF THE LUNG

GEOFFREY FLAVELL

CYSTIC DISEASE of the lung may be congenital in origin, or it may be acquired The distinction is often hard to make since pathological changes in the lung soon after birth may be mistaken for, or indistinguishable from, truly congenital lesions; and defects first seen in later life may have their root in developmental faults. Degenerative changes, certain specific pneumonias, the tension cavities of pulmonary tuberculosis, bronchial obstruction by neoplasm, secondary epithelialization of inflammatory spaces and parasitic infestation can on occasion all produce similar appearances Some are truly cysts, some abscesses, and some mere tears in the parenchyma modified by the elastic tensions of the web in which they occur The differential diagnosis between these lesions, often identical both in their clinical manifestations and their x-ray appearances, presents a task of exceptional difficulty which may be soluble only by histological means On the other hand the history, clinical course, and allied features of many pseudo-cysts will satisfactorily identify them

Tuberculous cavitation, for which cysts have commonly been mistaken, should be revealed for what it is by infiltration elsewhere and the finding of bacilli; pyogenic acute abscesses are generally associated with some obvious cause, and when they appear to lack this, or their clinical behaviour is atypical, they demand close investigation , those secondary to neoplasm may either fall into this latter group, or are clearly the product of an obstructing or necrotic growth But multiple cysts may so closely resemble cystic bronchiectasis that opinion is still divided as to whether bronchiectasis is not sometimes congenital, though most cases seen in infancy are now believed to follow early atelectasis When cysts are full of fluid there is practically no way of distinguishing them from any of the solid pulmonary tumours, or indeed sometimes from localized effusions or empyemas Finally a division has to be made between the cysts of congenital, and mainly bronchial, origin and those which are acquired, and arise from the alveoli

The symptoms accompanying cysts are largely the symptoms of their complications but successful treatment depends wholly upon the diagnosis of a cyst as prime cause, and failure to make this diagnosis means that treatment, too, must fail

CONGENITAL CYSTS

When the developing lung buds grow out from the primitive fore-gut of the embryo they may be disordered at any point along their course If this occurs at an early stage a fragment may be pinched off near the dividing trachea and a mediastinal bronchial cyst be left , while if a more substantial part of the anlage goes astray a whole sequestrated pulmonary lobe results Further peripherally, cysts will be found in the lung, solitary if one bronchial bud is involved, multiple if many are

involved, if a bronchial communication is present the cysts will also contain air and be subjected to the changes in its pressure, but, if isolated, they may be full only of fluid or of mucus which may gradually distend them or reach a state of quiescent equilibrium

In all cases these cysts are characterized by a lining of respiratory epithelium, cuboid or columnar, ciliated or not, as well as by a wall in which the elements of a bronchial wall, fibrous and elastic tissue, mucus glands and fragments of cartilage, are present, usually in disorderly array It is the possession of these features which principally distinguishes them from other cysts of the lung Kindred anomalies such as conjoined and supernumerary ribs, but particularly aberrant pulmonary vessels and unusual segmental fissures, are frequent and are strong evidence of congenital disorder

Large cysts are detected in early infancy because they make their presence felt They are generally air-containing, and when the first breaths enter the newly expanded lung it balloons in such a fashion that the small and pliant bronchus to which they are connected is compressed against the bulging cyst wall and its entry made more and more oblique, until at last air is admitted only during the bronchial expansion of inspiration and can no longer escape with contraction and expiry A vicious circle is thus established and the cyst grows with every breath and cry At first only the related lung is compressed, but soon the cyst fills the hemithorax causing respiratory distress and cyanosis, each growing more severe with every gasp until the mediastinum is displaced, and carries with it the trachea and the heart At this stage all the signs and symptoms of a tension pneumothorax will be present, x-rays show a deviated mediastinum, the functioning lung of one side compressed by it, while upon the other is no sign of pulmonary markings, the cyst itself conforming completely to the chest wall This predicament in infancy is practically always due to a ballooning cyst, and the insertion of a tension pneumothorax needle high in the chest, connected by tubing to a water seal, is lifesaving Puncture of the cyst may cause pneumothorax, and then the delicate arc of the cyst wall is sometimes visible on x ray examination, making the diagnosis plain, since air entry into the cyst is valvular, however, such pneumothoraces afford no relief but are in turn subjected to an increasing positive pressure

Solitary fluid-containing cysts, or air cysts in which a check valve mechanism has not developed will, if large enough, cause pressure symptoms by their bulk alone, but should this not be the case they lie fallow in the lung probably about the size of oranges, until overtaken by infection (Fig 132) Multiple cysts, similar in their behaviour and in histology are in general smaller, and either cluster in one lobe like a group of two or three soap bubbles, or involve an entire lobe or even lung in diffuse honeycombing amongst which almost no parenchymal tissue survives While this may resemble acquired "cystic" bronchiectasis, peri bronchial inflammatory damage is wanting, the cysts are bigger than usually occurs in bronchiectatic dilatation, and other stigmata of congenital disturbance are often present (Fig 133) Such silent cysts will continue undetected into childhood, adolescence or early adult life, although attention may be drawn to them by an unexplained, persistent cough, dyspnoea or haemoptysis The latter is more often found when cystic disease is diffuse, and is occasionally severe Asthma or early



FIG 132 (*a*) and (*b*).—A typical congenital bronchial cyst communicating with a bronchus The patient had no symptoms and the cyst has not yet become infected so that the fluid in its lower third is probably mucus

(*a*)



(*b*)

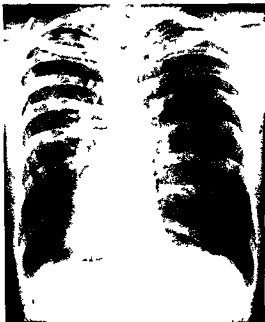


FIG 133—A case of cystic honeycombing involving the whole of the right upper lobe and associated with dextrocardia This combination is sometimes known as Kartagena s syndrome The congenital malposition of the heart is convincing evidence of the congenital origin of the cystic disease in the upper lobe

clubbing in children especially if they appear somewhat undersized, and if the chest seems curiously deep or asymmetrical should indicate the possibility that congenital cysts are present The right lung is more commonly affected than the left

Sooner or later since the great majority communicate with the bronchial tree lung cysts are likely to become infected If they are single chronic ill health with cough recurrent fevers and purulent sputum may follow and should x ray examination reveal a fluid level in the chest lung abscess or empyema is perhaps diagnosed It has often been the case that large parietal fluid-containing cysts even when not infected have been mistaken for empyemas and drained by rib resection indeed ill advised transpleural aspiration of an infected cyst is calculated to result in true empyema Drainage however does not result in the progressive diminution of the cavity and a long period of illness with a persistently draining sinus will continue until it is realized that a cyst is present and resection carried out Infected cysts may also rupture into the pleura or suddenly establish a fistulous communication with the bronchial tree flooding it with pus Where

cerebral abscess is ever present

Islands of pulmonary tissue having no connexion with the adult bronchial tree or at most a tenuous one are sometimes found in the chest They receive no blood

supply from the pulmonary circulation, but are nourished by an aberrant vessel rising directly from the aorta, or the coeliac plexus (in which case it pierces the diaphragm from below), and representing one of the foetal vascular plexuses Analogous in origin both to bronchial and enterogenous cysts, since they are the result of an abnormal and isolated outgrowth from the primitive fore-gut, such pulmonary islands are commonly situated posteriorly in the left chest (Fig 134) They are sometimes at the apex, where they may have a narrow connexion with the side of the trachea, or wholly sequestrated at the base ; and they cast upon x-rays the opaque shadow of airless lung It is not surprising, in view of their origin, that they are on occasion partially or wholly cystic, and that such cysts may become secondarily infected *Bronchography shows a normal bronchial tree having no* connexions with the sequestrated area At thoracotomy they are adherent to surrounding tissues, and the artery supplying them may not only be unexpected in position, but also of formidable size

Treatment

The correct treatment of congenital cysts, whether they be the ballooning air [illegible]

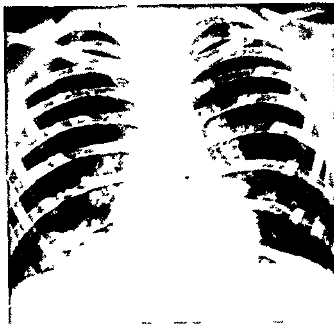
[illegible] the case of honeycombed lobes or lung, lobectomy [illegible] tances the preservation of [illegible] the surgeon's aim

ACQUIRED CYSTS

The cysts of this category, with the exception [illegible] 229), are pseudo cysts, inasmuch as they lac[illegible] cysts, and walls peculiar to themselves, if [illegible] bronchiectasis and other pulmonary cavities of specific provenance are likewise set on one side, those remaining share also the characteristic that they arise from the alveolar part of the lung and not from the bronchi, and that they always contain air

Blebs and bullae

[illegible] develop under [illegible] ure the normal [illegible] in the alveolar mesh, smooth rounded cavities appear surrounded by a narrow atelectatic zone, and grow in size disproportionately to the pressure exerted upon them (Moolten 1935) Not only is the lung itself an elastic net, but it is subjected [illegible] and as a hole [illegible] gap in the [illegible] y coalesce [illegible] m If the [illegible] ht degree,



(*a*)



F G 134 (*a* and *b*) —A seques trated lobe w th marked cys c changes s tuated on the pos ero- nfer or aspect of he r ght lower lobe in a ch ld of 6 years Numerous fistulae ex sted between it and the base of the r ght lower lobe through wh ch a great deal of pus dra ned It ob a ned i s blood supply through a large arter al trunk p erc ng the r ght d aphragm f om below At operat on t was poss ble to cut it bod ly from the ba e of the lower lobe w hout bleed ng The fistulae were closed and the ch ld made an un n er rupted reco ery

(*b*)

air may be able to enter the alveoli supplied by it during the bronchial dilatation of inspiration, but not to escape again when contraction occurs with expiration Thus a small alveolar space may rapidly balloon (the effect being enhanced by the *forced inspirations that precede coughing) and the surrounding parenchyma* compressed before the advancing bubble Ultimately it may reach an equilibrium, or it may continue to expand until it bursts through the visceral pleura, producing a pneumothorax , or grow still vaster as ballooning congenital cysts do, producing mediastinal displacement and symptoms of compression

A similar mechanism is well seen *in the tension cavities that accompany tuber*culosis, and it is perhaps significant to observe that remarkable thin-walled cystic cavities often appear during the course of acute staphylococcal pneumonias *These cavities are at first quite empty, but later have a level of staphylococcal* pus in them (by which alone they are to be identified as abscesses) and, now aided by antibiotics, shrink and vanish as quickly as they came (Fig 135) It seems *probable that some at least of the ballooning cysts seen in children, when alveolar* and not bronchial in type, may be " cysts " of this sort, secondary to an unrecognized staphylococcal pneumonitis, rather than cysts of congenital origin , more *especially since the sudden onset of staphylococcal empyema in infancy was once* common and could be explained by the same process

The pneumatocoeles known as blebs and bullae appear first as a localized breakdown of alveolar walls weakened by infection or by the degenerative changes of chronic emphysema The vesicle is then extended by the inherent stresses of the lung, and finally blown up by a valvular ingress of air Blebs are defined as subpleural collections of air caused by rupture of alveoli immediately beneath the visceral pleura , while bullae result from the coalescence of one alveolus with [illegible]

and stresses [illegible]

earlier age-groups is obscure but may depend upon inflammatory [illegible] terminal bronchioles by pneumonitis or, it has been suggested, by healed sub*clinical pulmonary tuberculosis It is also conceivable that they might arise* because of a congenital defect in the bronchial wall, but this seems less likely

In infants they may attain great size, collapsing the lung from which they spring [illegible] *the mediastinum in the same manner as ballooning congenital cysts,* [illegible]

gressive respiratory distress with *cyanosis* [illegible] from them produces either no effect or results in pneumothorax Occasionally they vanish as mysteriously as they appear, resembling in this respect the thin-walled " abscesses " of *staphylococcal pneumonia Radiologically there is com*plete absence of lung markings on the affected side, but fine trabeculae may be



FIG. 135 (*a* and *b*).—Acute staphylococcal abscesses in the lung simulating cysts

(*a*)

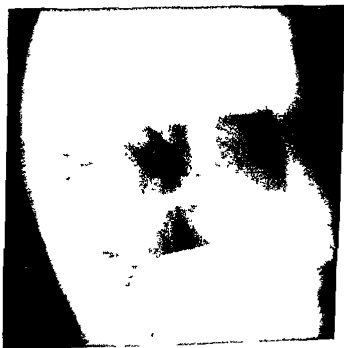


(*b*)



(a)

FIG 136 (a and b) —A ballooning lung cyst in an adult patient There is no evidence of emphysema in the rest of the lung fields



(b)

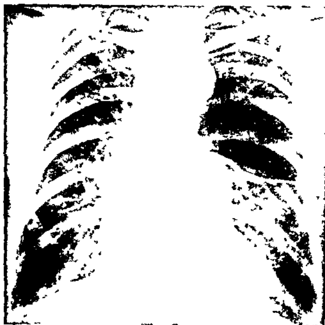


FIG 137 —(*a*) A giant cyst related to the left upper lobe in a man aged 66 years and simulating a spontaneous pneumothorax At operation it was excised and fistulous communications with the compressed upper lobe were sutured (*b*) the lung then re expanded without further difficulty to fill the chest

(*a*)



(*b*)

distinguishable especially at the base of the cyst or the curvature of its edge seen in the costo phrenic angle

During the second and third decades of life similar blebs and bullae occur but do not often reach the large proportions of infancy The lungs show no other sign of disease and are not generally *emphysematous, the cysts being confined to* one area While they may cause dyspnoea by progressive enlargement their chief importance at this age lies in their being the source of idiopathic or asymptomatic spontaneous pneumothorax A high proportion of such pneumothoraces are chronic the lung not re expanding within three months and frequently recurrent They thus result in severe incapacitation often unnecessarily prolonged by inadequate or ill conceived treatment and carry as well a serious risk to life since an infection or similar accident upon the opposite side is likely to cause death Inspection of the lung with a thoracoscope may reveal the collapsed and punctured bleb but sometimes this is not apparent and must be searched for at thoracotomy like a leak in a bicycle tyre

In the past such pneumothoraces have been treated by inducing chemical pleurisy with insufflated iodized talc or silver nitrate among many other irritants and withdrawing air in the hope that the lung will adhere to the chest wall The process is painful not always successful and tends permanently to diminish respiratory efficiency There is little doubt that in most cases and certainly in *all those where the lung fails to re expand within a few weeks direct attack upon* the cyst provides the best hope of early and lasting cure Even though the patient may have a seriously reduced respiratory capacity either because of a large cyst or because of a pneumothorax as soon as the chest is opened and the bleb punctured or the leak secured it is possible to re expand the lung and restore normal respiration *These cysts can be separated from the neighbouring tissue by sharp* dissection The interior is often multilocular being divided by trabeculations which are characteristic when seen in x ray films and at the base are usually a number of small bronchial communications requiring closure *Such operations* promptly carried out save the patient months of illness and distress as *well as* avoiding the mortal dangers of contra lateral disease

The bullae seen in older men and associated with emphysema fortunately do not have this tendency to burst perhaps because their connexions with the

None-

become marked local excision of the bulla allows [illegible]

but other bullae will undoubtedly be present in the lung probably bilaterally and each case must be judged upon its merits and necessity

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CHAPTER 25

TUMOURS OF THE LUNG

VERNON C THOMPSON

THE ORIGIN *of a pulmonary tumour is almost always from the cellular structures of a* bronchus Far the most common tumours are those arising from the bronchial epithelium, and in this category carcinoma comprises about 98 per cent

Although benign tumours of the lung are comparative rarities their clinical recognition is of the utmost importance If they are diagnosed and treated the *prognosis for the patient is excellent if undiagnosed they may cause death by their* complications or they may produce years of invalidism

The following classification is offered as a basis for further study *It cannot claim to be complete as it omits such rarities as myoma endothelioma and other* pathological curiosities that have been reported from time to time , but it includes *the lesions of main clinical importance, in a natural order of precedence*

Primary malignant	Carcinoma
	Sarcoma
	(a) round celled sarcoma
	(b) fibro sarcoma
	(c) lympho sarcoma
Secondary malignant	Carcinoma, sarcoma, etcetera
Benign	Adenoma
	Hamartoma
	(a) chondroma
	(b) fibroma
	(c) lipoma
	(d) vascular hamartoma
	Neurogenic tumours

PRIMARY CARCINOMA OF THE LUNG

Incidence and aetiology

... the sexes ... the incidence of primary ... on in males as ... from this age ... mum incidence ... cline Mason (1949) analysing 1 000 cases finds the incidence of age and percentage as follows 30 years, 2 2 per cent 30–40 9 6 40–50, 30 9 50–60, 37 6 , 60–70, 18 6, over 70, 1 1 These figures may be taken to represent common experience in any large series of cases There is no doubt that there has been a steady increase in the incidence of cancer of the lung in both sexes in the past 50 years Hutchison (1928) drew attention to the increasing incidence of this condition in the first quarter of the

century. In the second quarter the increased incidence has far exceeded that of the first. In 1922 the number of deaths from this cause in England and Wales was 612, a fairly insignificant number. In 1949 cancer of the lung accounted for 11,272 deaths. In males the lung has now become the commonest site of primary malignant disease.

The cause of this increase has been a matter of considerable speculation and investigation. Speculation suggests that the increase may be accounted for by improved methods of diagnosis, and the survival of more people into the cancer age. These factors must undoubtedly account for at least some part of the supposed increase in incidence, but they can hardly account for the whole. Investigation has mainly been devoted to the search for some inhaled carcinogen which has increased *pari passu* with the increasing incidence of carcinoma of the lung.

The possibility of an inhaled carcinogen has been recognized for many years, and the increased incidence of carcinoma of the lung among workers in certain occupations has been studied throughout the years. The most noted example of this disease as an industrial hazard is among workers in the Schneeberg mines in Czechoslovakia. In more recent years attention has been drawn to the added risks of cancer of the lung in workers with certain mineral ores. Perry (1947) has drawn attention to the increased incidence among workers in arsenic and in certain nickel and copper ores in which arsenic occurs as an impurity. Machle and Gregorius (1948) have reported that the death rate for cancer of the lung was 25 times the normal among workers in the chromate-producing industry in the United States of America.

It is notable that there is little or no evidence that chronic irritation by inflammation such as occurs in chronic bronchitis, pulmonary tuberculosis or bronchiectasis has any specific bearing on the development of carcinoma, though most clinicians of wide experience are able to recall isolated examples of the development of neoplasms in chronically inflamed lungs. No definite association has been found between carcinoma and the inhalation of dust in general, as typified by coal dust producing anthracosis in miners.

Wynder and Graham (1950) in the United States of America and Doll and Hill (1950) in Great Britain report statistical investigations into the effect of tobacco smoking on the incidence of carcinoma of the lung. They point out the correlation between the increase in the habit of cigarette smoking and the increase of lung cancer. They each report a careful review of the smoking habits of a large series of patients with carcinoma of the lung as compared with a similar number of controls. Their conclusions that heavy cigarette smokers run a far greater risk of lung cancer than non-smokers are irrefutable. Doll and Hill go so far as to say that the risk of developing the disease increases in simple proportion with the amount smoked, and after the age of 45 the risk in heavy smokers may be as much as 50 times as great as among non-smokers.

The evidence so far suggests that though chronic inflammation and irritation by non specific dusts are not entirely innocuous, the development of carcinoma of the lung is much more probable when specific carcinogens are inhaled. These carcinogens may occur in dusts or fumes associated with certain occupations and may occur in tobacco smoke. The precise nature of the carcinogens has not been

determined with any certainty Much work has been done on this subject, but much more remains to be done in further elucidation of the problem

Pathology

Histology

There was considerable confusion about the histology and nomenclature of carcinoma of the lung until Barnard (1926) following Turnbull and Bartlett showed that malignant epithelial tumours of the lung were often pleomorphic, but all could be derived from the basement layer of the bronchial mucous membrane Before this date many of the primary lung tumours had been classified as "oat cell sarcoma" or "lympho-sarcoma" of the mediastinum

Perhaps the commonest histological type of lung carcinoma is the squamous celled growth which evolves with metaplasia, for squamous epithelium is not a normal component of the bronchus For purposes of description carcinoma of the bronchus is divided into three types

(1) The oat-cell carcinoma, so called in Great Britain (Fig 138) This term is employed because the cells resemble oat seeds, they have large darkly staining nuclei and little cytoplasm They are in general undifferentiated tumours with little fibrous stroma In other countries they are usually referred to as small-celled or anaplastic growths

(2) The squamous-cell carcinoma (Fig 139), composed of flattened squamous cells with a varying degree of fibrous stroma often showing keratinization In the more highly differentiated types cell nests are present

(3) The columnar-cell carcinoma (Fig 140) in which the growth is composed of well-established columnar cells which have an alveolar formation with well-developed stroma There may be intra-alveolar papilliferous projections About one half of the columnar-cell tumours secrete mucus

Although these three main types are described, it is not always possible in any particular case to place a tumour in any one of these categories, for different types of cells may be found in the same tumour, and transitional cells such as spheroidal cells and polygonal cells are encountered The classification of a group of tumours will therefore always depend to some extent on the personal opinion of the pathologist

The incidence of oat cell and squamous cell tumours is about equal The least common is the columnar-cell tumour which comprises only about 10 per cent of the total

Altho[illegible]gh there must be innumerable exceptions to any generalization there are [illegible]

When it occurs in a major bronchus it metastasizes early to the lymph nodes which become tense with growth and the pericardium is involved early (Fig 141) Squamous cell carcinoma is more common in elderly people, it varies greatly in its malignancy It may grow very slowly and metastasize late It occurs, like all lung tumours, with greatest frequency in the major bronchi, but when it develops peripherally it shows a tendency to break down in its centre and cavitate (Fig 142)

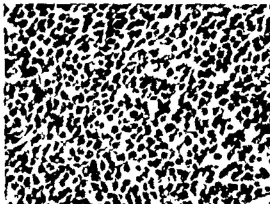


FIG 138—*Oat-cell* carcinoma (× 470 Reduced one quarter on reproduction)

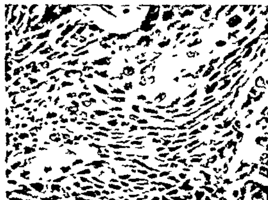


FIG 139—*Squamous-cell* carcinoma (× 330 Reduced one quarter on reproduction)



FIG 140—*Columnar-cell* carcinoma (× 210 Reduced one quarter on reproduction)

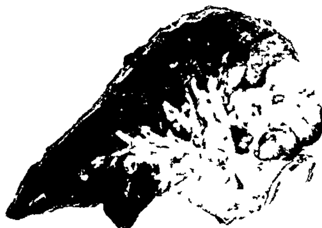


FIG 141 —Oat-cell carcinoma of lung showing mediastinal extension involving and surrounding great vessels Autopsy specimen



FIG 142 —Squamous-cell carcinoma of upper lobe showing necrotic growth with cavitation simulating lung abscess Operation specimen

In these cases it may extend centrifugally and involve the chest wall before metastasizing The adenocarcinoma is remarkable chiefly for its incidence Although all other types are far commoner in men than women, adenocarcinoma is almost equally divided between sexes It shows one other characteristic which is somewhat exceptional This is its tendency to spread by seedlings once it involves the pleura the latter rapidly becomes coated with small seedlings which coalesce and produce plaques of growth It is this tendency to pleural spread which has often masked the primary tumour and caused the erroneous diagnosis of endothelioma of the pleura Adenocarcinoma also tends to seed itself bronchogenically *so that a small primary tumour may produce miliary bronchogenic seedlings* throughout the lung

Macroscopic appearance

About 75 per cent of all lung tumours arise in the major bronchi 25 per cent arise in the smaller bronchi and are situated peripherally in the lung Seldom do they occur in the trachea Those occurring in the major bronchi may present as proliferating endobronchial tumours (Fig 143) or they may produce a funnel shaped stricture by sub mucous infiltration (Fig 144) Eventually they cause obstruction of the bronchus with collapse of the alveoli distal to the growth This



Fig 143—Adenocarcinoma of lung showing intra bronchial proliferating growth from parenchymatous mass in right upper lobe Operation specimen



Fig 144 Squamous cell carcinoma showing peri bronchial infiltration and stricture of the bronchus Operation specimen

collapse may be segmental lobar or total according to the site of the primary tumour Secondary changes in the lung occur distal to the growth Even in the absence of infection mucus distension and bronchiectasis appear early If there is infection suppurative pneumonitis bronchiectasis and lung abscess may complicate the condition These complications may extend to the pleura so that pleural effusion or empyema may ensue Growths situated peripherally are more likely to present in the early stages as solid rounded tumours without the complications of bronchial obstruction but as they extend they involve structures outside the lung

Mode of spread

A malignant tumour of the lung may spread in many ways

Direct extension

In tumours near the hilum this extension tends to be towards the mediastinum so that the great vessels and the pericardium and the phrenic and vagus nerves become involved. In peripheral growths the extension may reach the chest wall without other complications causing erosion of ribs and spreading through the thoracic cage into the soft tissues beyond. At the apex of the lung the brachial plexus, the sympathetic chain, the nerve roots and the vertebral column may become involved. At the base of the lung the primary growth may infiltrate the diaphragm.

Metastasis

Metastasis may be by the lymphatics, by the blood stream or by the bronchi.

Lymphatic spread

Lymphatic spread occurs as a rule first to the neighbouring lymph nodes at the hilum of the lung; thence the growth spreads into the mediastinal lymph nodes where it may cross the mid-line and extend upwards into the neck and downwards to the retroperitoneal lymphatics below the diaphragm.

Another form of lymphatic spread may occur in the lung itself by a process of linear permeation, producing the condition known as carcinomatosis lymphangeosa (Fig. 145).

Blood-borne metastases

Dissemination into the blood stream occurs as a result of the escape of emboli into the pulmonary veins. As might be expected these emboli are often carried into the cerebral vessels and cause metastases in the brain; sometimes these metastases are solitary. Once an embolus escapes into a pulmonary vein it passes into the systemic circulation and may lodge and grow anywhere in the body—usually haematogenous metastases are multiple. In addition to the brain they show a particular predilection for the liver, the bones, the suprarenals and the skin; but no tissue is immune (Fig. 146).

Bronchogenic metastasis

Rarely, some carcinomas tend to grow like a fern into and within the lumina of the bronchi; this is a method of direct spread. From this primary growth seedlings may become separated and become dispersed into other parts of the bronchial tree where they proceed to grow separately. Usually these bronchogenic metastases occur in the same lung, but they may arise in the other lung. This particular form of spread is most often encountered with the columnar-cell growths.

Diagnosis

It is often a simple matter to diagnose advanced inoperable malignant disease of the lung. This diagnosis may be of some interest to the clinician; but the accuracy of the diagnosis is of little importance to the patient for by then his fate is sealed and he might just as well be suffering from any other rapidly progressive lethal condition. To diagnose tumours of the lung at an early stage is of supreme importance to the patient; and this can only be achieved by first suspecting the presence of a tumour and then carrying out a series of investigations to prove it.



FIG 145.—Carcinomatosis lymphangeosa

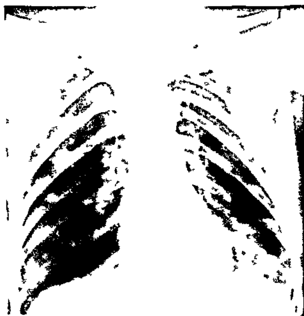


FIG 146.—Carcinoma of the left lower lobe with metastases throughout both lungs.

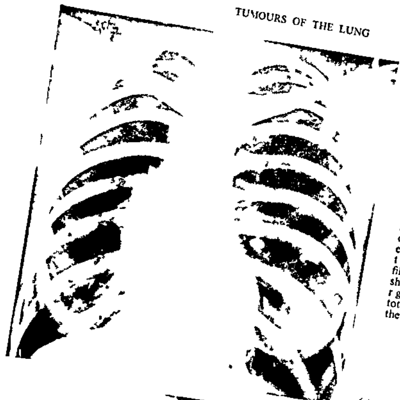
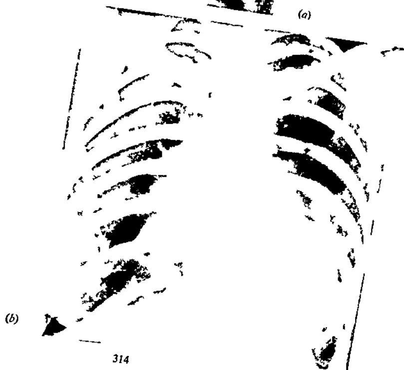


FIG 147—(a) A h lar mass in a young woman due to oat cell carc noma (b) film taken on exp ra tion shows med ast nal displacement away from the lesion due to obstruct ve emphysema by par t al obstruction (c) film two weeks later shows collapse of the r ght lung due to total obstruct on of the bronchus



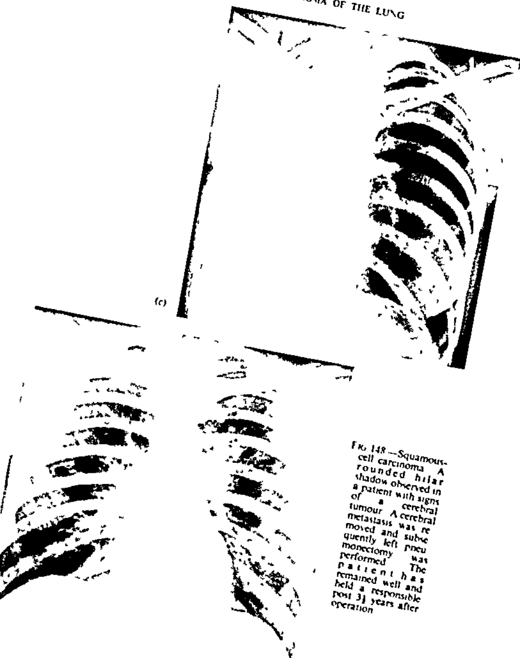
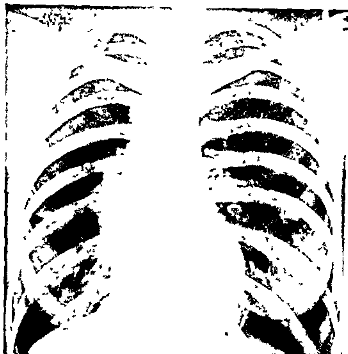


FIG. 148.—Squamous-cell carcinoma. A rounded hilar shadow observed in a patient with signs of a cerebral tumour. A cerebral metastasis was removed and subsequently left pneumonectomy was performed. The patient has remained well and held a responsible post 3½ years after operation.



(a)

FIG 147—(a) A hilar mass in a young woman, due to oat-cell carcinoma, (b) film taken on expiration shows mediastinal displacement away from the lesion due to obstructive emphysema by partial obstruction, (c) film two weeks later shows collapse of the right lung due to total obstruction of the bronchus



(b)



F G 148 —Squamous cell carc noma A rounded h lar shadow observed n a pat ent w th s gns of a cerebral tumour A cerebral metastas s was re mo ed and subsequently left pneumonectomy was performed The pat ent has rema ned well and held a respons ble post 3½ years after operat on

Symptomatology

It is unfortunately well substantiated by mass radiography, and chance findings on screening the chest at radiological examination for other lesions such as peptic ulcer, that carcinoma of the lung can be quite silent and symptomless until its intra-thoracic extensions have rendered it inoperable It is also not uncommon for the first symptom to be caused by metastases Before resection of the lung became a safe and successful procedure, the outlook for patients with pulmonary neoplasms was indeed hopeless and there was little to be gained by making either an early or an accurate diagnosis In spite of the prospects of successful surgery in certain cases the prognosis is still grave, partly because of the silent onset of the disease and partly because of the tendency to metastasize The outlook however is not so grave as to justify the pessimistic and fatalistic attitude that is still prevalent and which perhaps accounts for the long delay in diagnosis that occurs even when symptoms are present Bjork (1947) analysing 112 inoperable cases found an average delay of 8 4 months from the first symptom to reference to hospital Mason (1949) found the average duration of symptoms in his 1,000 cases was over 6 months In the writer's experience at the London Hospital the average duration of symptoms is 7 months

Cough is the commonest first symptom—this is such a prevalent malady, particularly in the winter, that it cannot be fully investigated at once , but when it occurs in a patient over 40 and persists without adequate reason for more than 2 or 3 weeks a fuller investigation is indicated The other more common early symptoms are chest pain, dyspnoea haemoptysis and lassitude Haemoptysis is *a fairly dramatic event and should never be treated expectantly without proceeding* to a diagnosis unfortunately it is not the commonest first symptom Brooks and
[illegible] 14 22 per cent of 502

The symptomatology of carcinoma of the lung can only be fully understood by reference to its pathology When the growth is peripheral it is silent until it breaks *down or ulcerates into a major bronchus, then it may* cause cough, haemoptysis or purulent sputum from secondary infection If it extends to the pleura it causes chest pain , or it may announce itself by metastases

When the primary growth arises in a major bronchus it causes cough, possibly haemoptysis As it encroaches on the lumen of the bronchus it may cause a persistent wheeze and dyspnoea by obstruction of the air-way Later there may be obstructive emphysema with increase of breathlessness This is followed when the obstruction of the bronchus is complete by collapse of the lung distal to the growth and complications due to infection ensue (Fig 147) The symptoms may then become those of pneumonia, bronchiectasis, lung abscess, pleurisy or empyema Loss of weight should never be regarded as a pure symptom of a primary growth *of the lung when it occurs in association* it is usually due to toxaemia caused either by metastasis or infection

The mode of onset of symptoms in carcinoma of the lung may be so diverse that it is possible to describe certain quite different clinical types of onset In a large

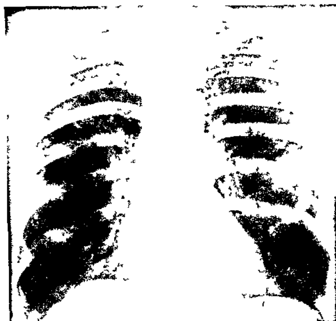


Fig 149 —Peripheral squamous-cell carcinoma A small round shadow in a patient aged 60 years who presented with no chest symptoms but with marked pulmonary osteoarthropathy Treated by left lower lobectomy with immediate relief of joint pains and no evidence of recurrence 3 years later

hospital with special departments patients may be referred on their first attendance to many different divisions of the hospital The Department of Chest Disease inevitably invites the majority of cases because of their frank pulmonary symptoms The Department of Cardiology sees a number of patients who are referred for dyspnoea , and sometimes patients are referred with paroxysmal cardiac irregularities which are due to involvement of the heart itself by the growth Cases of mediastinal obstruction and superior vena cava obstruction also find their way to this department The Departments of Neurology and Neurosurgery see a number of patients with primary carcinoma of the lung Taylor and Waterhouse (1950) found that the first symptom was neurological in 6 1 per cent of their cases These patients fall into three categories , they are mostly those with signs of a cerebral tumour and metastases (Fig 148) The second [illegible]

patients complaining of hoarseness due to paralysis of the left vocal cord by involvement of the recurrent laryngeal nerve The Orthopaedic Department sees patients with bone and joint pains or spontaneous fracture due to metastases The Department of Physical Medicine may receive patients complaining of generalized joint pains described as rheumatism, which are actually due to pulmonary osteoarthropathy (Fig 149) The Department of General Medicine

which always treats a number of cases of frank chest disease will also find patients with carcinoma of the lung referred for pyrexia, toxaemia or cachexia without dominant chest symptoms *This department will also find a small number of* cases among those referred for abdominal symptoms

Physical examination

There are no physical signs which are absolutely conclusive in diagnosing carcinoma of the lung except those of inoperability In early cases no abnormality whatever may be detected on ordinary clinical examination

The physical signs in the chest itself are those of the varied lung pathology which may be caused by *the tumour and its complications Impaired air entry and a* persistent rhonchus may be caused by partial bronchial obstruction , later there may be signs of pulmonary collapse with shift of the trachea or apex beat towards the side of the lesion and impaired breath sounds and impaired resonance to percussion over the collapsed portion of the lung (Fig 150) Obstructive emphysema may cause *hyper-resonance and almost absent air entry when the bronchus is* obstructed only on expiration

[illegible]

to percussion and displacement of the mediastinal structures away from the growth

The physical signs of the lung lesion may be marked by the signs of pleural *effusion When pleural effusion is associated with underlying pulmonary* atelectasis the displacement of the mediastinum that might be expected as a result of the fluid is counteracted by the opposite pull of the collapsed lung

The general examination of the patient must be thorough It will not help in diagnosing the early case , but it is essential to correct treatment and may reveal *metastases that contra-indicate operation No part of the body is quite immune* from metastases Special attention should be paid to the nervous system, the supraclavicular lymph nodes, the skeletal system, the liver and the skin

Fever and cachexia are signs of secondary infection or metastases The time to diagnose carcinoma of the lung is when the patient is still in good health, not when "*he looks like a case of cancer*"

Special investigations

[illegible] story, the
[illegible] e com-
[illegible] ake or

exclude a positive diagnosis of growth

Radiographic examination

Tumours arising in a major bronchus cannot be revealed by a plain x-ray until they cast a shadow either by their own bulk or they produce complications such as pneumonia or atelectasis If obstructive emphysema is suspected clinically, it should be clearly indicated to the radiologist This condition may not be shown in the normal film taken on inspiration, but it becomes quite obvious if films are taken *on expiration as well* (Fig 151)

FIG 150—(*a*) An indefinite shadow below the left hilum due to an anaplastic *carcinoma of the* lower lobe (*b*) film taken three weeks later showing collapse of the left lower lobe with a small pleural effusion

(*a*)

(*b*)

With the proviso that early growths cannot be disclosed by routine chest films, it is true to say that sooner or later the growth can be discovered by radiology In actual practice the great majority of growths of the lung are detectable by x ray examination by the time the patient seeks medical advice The radiological picture is either one of pulmonary obstruction and its sequelae of collapse and infection (Figs 152 and 153), or the opacity seen in the radiograph is caused by the mass itself, in which case the tumour is usually peripheral The peripheral growths may appear as solid rounded or lobulated shadows, often with an ill defined edge with radiating striae extending from it, or they may show a cavity with a fluid level due to necrosis in the substance of the shadow (Fig 154) Mason (1949) found that in 64 per cent of cases the picture was one of collapse, and in about 10 per cent a cavity mimicking a chronic lung abscess was seen

Bronchoscopy

a simple and harmless procedure, tumours involving the major bronchi are seen directly and a biopsy can be taken In other cases though a biopsy may not be possible the diagnosis may be inferred from narrowing, rigidity and distortion of the bronchial tree, when these findings are associated with the radiological appearances Apart from diagnosis bronchoscopy is an essential investigation in determining the operability and the correct treatment of pulmonary tumours

Only a limited part of the bronchial tree is visible on bronchoscopy, so negative findings do not necessarily exclude a diagnosis of carcinoma

Sputum examination

Sputum examination is not an important matter in establishing the diagnosis when the bronchoscopic findings and particularly the biopsy are positive, but when bronchoscopy is negative, examination of the sputum for malignant cells is of considerable value

which cannot be excluded from any well organized chest unit, depends entirely on the individual skill and experience of the pathologist who examines the sputum When bronchoscopy gives no positive evidence sputum examination may reveal malignant cells in those cases of peripheral growth in which there is central necrosis and ulceration but sputum examination may still be negative if the growth is peripheral and symptomless

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segmental filling defect by bronchography, but this is usually radiography which demonstrates the pulmonary segmental collapse distal to the obstruction

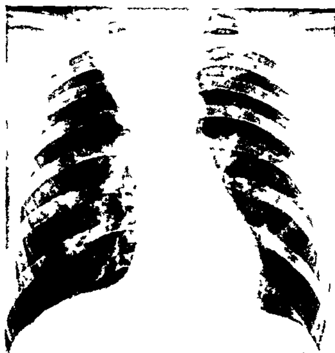


FIG 151 —Obstructive emphysema due to carcinoma of the left main bronchus (*a*) on inspiration shows no gross abnormality though there is mediastinal displacement to the left and some increase in the left hilar shadow (*b*) taken in expiration shows failure of the air to escape from the left lung with mediastinal displacement to the right On physical examination there was no air entry to the left lung The patient was breathless

(*a*)



(*b*)



(a)

Fig 152—Collapse of right upper lobe due to carcinoma of bronchus treated by pneumonectomy (a) before operation (b) 2 years after pneumonectomy



(b)

If serial skiagrams show that a shadow in the lung field is growing steadily in size it is almost certainly a growth

A wedge shaped opacity corresponding anatomically with a pulmonary segment is due to bronchial obstruction and in patients over the age of 40 the most probable cause of the obstruction is neoplasm

The peripheral carcinoma that breaks down and presents radiographically as an abscess with fluid level can be differentiated from a chronic lung abscess by the character of its wall In neoplasms the wall is uneven and presents irregularities and bosses which are never present in a pure pyogenic abscess The clinical history in these two conditions is also characteristically different In chronic abscess there is usually a history of copious purulent sputum in neoplasm the sputum is less in amount and generally it is persistently blood stained

The solid tuberculous focus may present great difficulty in differential diagnosis As a rule tuberculous lesions in the lung are not solitary so the difficulty does not often arise The presence of tubercle bacilli in the sputum is confirmatory evidence of tuberculosis but this finding does not necessarily exclude a diagnosis of carcinoma for the two diseases may co exist A period of observation by repeated x ray examination is justifiable only if the clinician makes the reservation that the lesion should be regarded as a growth if it becomes larger

Treatment

The treatment of choice for carcinoma of the lung is resection In most cases total pneumonectomy is required For technical reasons alone it is usually necessary to remove the whole lung in order to remove the growth It is desirable when operating for malignant disease to perform a so called radical operation by excising the area of lymphatic drainage together with the tumour This type of operation can only be accomplished by total pneumonectomy Even so the operation cannot be regarded as a complete radical excision as the lymphatics transgress the midline and are not all accessible to the surgeon

The operation of lobectomy permits only a limited excision of lymphatics but it has a definite and valuable place in the treatment of lung cancer The loss of a whole lung may cause serious incapacity to a patient over the age of 60 the loss of a lobe produces little disability Patients with low cardio respiratory reserve may die of heart failure after total pneumonectomy or they may survive only to be crippled by dyspnoea The preservation of healthy lung tissue in such patients is often of more importance than the hypothetical value of a radical operation

The malignancy of primary neoplasms of the lung varies enormously and the no is after operation seems to depend more on the degree of malignancy than r d cal nature of the operation

ı tory statistical evidence exists as to the operabil ty of malignant

Tomography

In delineating the depth, the shape, *the edge and the relations of certain indefinite* shadows tomography has some value but it cannot give a positive diagnosis Tomography may be misleading when it is employed to demonstrate strictures of the bronchi none of the bronchi is quite parallel with the plane of the tomo graph and even when normal they may appear to become narrowed as they transect the level at which the film is focused

Aspiration biopsy

Aspiration biopsy and drill biopsy are mentioned only to b· condemned as normal methods of investigation These procedures should be employed only in cases in which it is clear from other investigations that the lesion is inoperable but almost certainly neoplastic If applied indiscriminately they may lead to complications such as pneumothorax, empyema and dissemination of the growth They are only of value in determining questions of prognosis and sensitivity to radiotherapy

Differential diagnosis

It is clear from the preceding sections that there must be a full investigation of patients suspected of pulmonary neoplasm and the steps in this investigation have been described There will remain a small proportion in whom the diag nosis will still remain in doubt These are the patients in whom the lesion is beyond bronchoscopic vision and radiology presents a persistent abnormality In this connexion it is important to consider the observations of Raeburn (1951) who regards bronchoscopic evidence as a fairly late sign of most pulmonary neoplasms Even so there are at present certain limits to our powers of diagnosis and we cannot claim to be able to diagnose carcinoma of the lung in the presence of normal x ray examination a normal bronchoscopy and a negative sputum examination

The differential diagnosis therefore limits itself to the interpretation of x ray shadows when bronchoscopy and sputum examination are negative It is of course possible that diagnosis may be delayed by failure to employ bronchoscopy and sputum examination because the radiological shadows resolve as a result of chemotherapy The antibiotic drugs are now so successful in resolving the infective complications of new growths that there is a serious danger that they may even further delay the diagnosis of the primary bronchial tumour Patients with atypical pneumonia of any sort that does not resolve symptomatically and radiologically within one month from the date of onset, should be suspected of [illegible] Most of the delays in diagnosis are due [illegible] cases [illegible] logical [illegible] scopy and sputum examination It is in this particular group that the skill the experience and the judgment of the clinician becomes of supreme importance The type of case that falls into this difficult category is the patient whose radiograph shows an atypical peripheral shadow It is beyond the scope of this chapter to discuss all the possibilities of diagnosis, but certain general principles may be laid down

If serial skiagrams show that a shadow in the lung field is growing steadily in size it is almost certainly a growth

A wedge shaped opacity corresponding anatomically with a pulmonary segment is due to bronchial obstruction and in patients over the age of 40 the most probable cause of the obstruction is neoplasm

The peripheral carcinoma that breaks down and presents radiographically as an abscess with fluid level can be differentiated from a chronic lung abscess by the character of its wall In neoplasms the wall is uneven and presents irregularities and bosses which are never present in a pure pyogenic abscess The clinical history in these two conditions is also characteristically different In chronic abscess there is usually a history of copious purulent sputum in neoplasm the sputum is less in amount and generally it is persistently blood stained

The solid tuberculous focus may present great difficulty in differential diagnosis As a rule tuberculous lesions in the lung are not solitary so the difficulty does not often arise The presence of tubercle bacilli in the sputum is confirmatory evidence of tuberculosis but this finding does not necessarily exclude a diagnosis of carcinoma for the two diseases may co exist A period of observation by repeated x ray examination is justifiable only if the clinician makes the reservation that the lesion should be regarded as a growth if it becomes larger

Treatment

The treatment of choice for carcinoma of the lung is resection In most cases total pneumonectomy is required For technical reasons alone it is usually necessary to remove the whole lung in order to remove the growth It is desirable when operating for malignant disease to perform a so-called radical operation by excising the area of lymphatic drainage together with the tumour This type of operation can only be accomplished by total pneumonectomy Even so the operation cannot be regarded as a complete radical excision as the lymphatics transgress the midline and are not all accessible to the surgeon

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The malignancy of primary neoplasms of the lung varies enormously and the prognosis after operation seems to depend more on the degree of malignancy than on the radical nature of the operation

Operability

No satisfactory statistical evidence exists as to the operability of malignant growths of the lung and it is logical to assume that there never can be any true assessment The operability of a tumour depends entirely on the date of diagnosis Our present methods of diagnosis are insufficiently employed Most authors are agreed that there is an average delay of 6–8 months from the date of the first

symptom until the patient comes to operation On the other hand mass radiography has revealed many patients without symptoms in whom the lesion is inoperable, and many more patients present with initial signs of inoperability such as metastases Although operability rates may be increased by more diligent and accurate methods of diagnosis, it seems inevitable that a large percentage of patients with carcinoma of the lung will be unsuitable for operation

Brock (1950) reports that of 800 cases seen in the years 1941–48 the operability rate was 13 25 per cent, of 449 cases seen 1945–48 the operability rate was 19 per cent showing an increase in operability rate in the later years These figures are probably higher than the general figure as the patients seen by an individual surgeon or in a surgical unit are preselected In general the operability rate is low, it reflects the failure to diagnose the condition at an early stage

The diagnosis of inoperability —During the routine examination and investigation attention should be directed not only to the diagnosis but also to the prospects for successful resection Advanced age accompanied by its normal degenerative changes such as myocardial weakness and pulmonary emphysema must be assessed in relation to the disease Age itself is not a contra-indication to operation, but there are few patients over 70 who have sufficient cardio-respiratory reserve to survive with one lung The main general contra indication to operation is dyspnoea Chronic nephritis, diabetes, hypertension and similar general conditions are not contra-indications to operations for cancer

The extension of the growth itself either by direct spread or by metastases is the usual sign of inoperability This extension should become evident in the investigation of the patient Particular attention should be paid to the central nervous system, the skin, the lymph nodes, particularly those in the supraclavicular region, and to the liver Pain which is not related to the actual site of the tumour may point to metastases in bone or even in skeletal muscles

Nerve involvement within the chest may affect the recurrent laryngeal nerve producing hoarseness by paralysis of a vocal cord Tumours at the apex of the lung may involve the brachial plexus and the sympathetic trunk, producing a *Horner's syndrome* and brachial neuralgia The phrenic nerve may be involved [illegible] of the neck and

trachea, or the growth may be seen to involve these structures directly

Pleural effusion is not always a sign of inoperability If the effusion is deeply blood stained it may be taken to denote pleural involvement, but if it is clear or even purulent the effusion may be the result of infection and in this event the lesion is not necessarily inoperable

Direct involvement of the chest wall as indicated by rib erosion is not a contra-indication to operation as it is often possible to remove a part of the chest wall *en masse* with the lung or lobe (Fig 155)

In spite of the most careful investigation there will remain a group of patients in whom operability can only be decided by thoracotomy When there is no absolute contra-indication the patient should be given the chance of survival that only surgery can offer At operation every effort should be made to remove the

FIG 155 —(*a*) A peripheral squamous cell carcinoma involving the chest wall and eroding the 4th rib The only symptom was pain (*b*) 3 years after left upper lobectomy at which the involved area of chest wall was excised *en masse* with the tumour

(*a*)

(*b*)

growth if this is technically possible Even if the operative findings point to early recurrence removal of the lung or lobe is the best palliative treatment once the chest has been opened Removal of the growth should include the mediastinal lymph nodes, but it may also involve resection of a part of the chest wall, the diaphragm or the pericardium and even *in exceptional cases a part of the wall of* the left auricle Some cases will defy the most heroic attempts at removal particularly when there is massive mediastinal involvement In the writer's experience of 232 operations for carcinoma of the lung in the period 1941–51, 27 per cent proved inoperable at thoracotomy

Results of resection

The first successful pneumonectomy for carcinoma of the lung was carried out by Graham in 1933 , this operation was the herald of the great advances that have been made in the surgery of malignant disease of the lung The technical difficulties of the operation and of anaesthesia have been overcome, so that now it is possible to remove a lung or a lobe with few technical complications Operative mortality depends not so much on the character of the operation as on the general state of health of the patient The antibiotics, particularly penicillin, *have contributed largely to safety Before the penicillin era healing of the bronchial* stump, which was almost invariably infected, was an uncertain feature of the operation and bronchial fistula was a common occurrence Infection of the remaining lung was also a serious contribution to morbidity and mortality In the writer's experience in the years 1941–45 the operative mortality was 28 per cent and the incidence of bronchial fistula was 48 per cent In the years from 1945–51 the operative mortality was reduced to 7 per cent and the incidence of bronchial

There are occasional examples of patients with growths of low grade malignancy who have survived for several years without resection of the tumour There are also innumerable examples of patients with tumours of high grade malignancy who have died of disseminated metastases in a few months or *even weeks If* patients survive for more than
for later recurrence to occur
first year after operation , thi
operation for advanced malignant disease Most of the patients for whom resection is employed as a palliative procedure die within 1 year The following Table shows the fate of 91 patients (80 pneumonectomies and 11 lobectomies) operated upon by the writer in the years 1941–48

Since 1948 the operative mortality has been reduced , but it is unlikely that the 3-year survival rate will be improved unless patients are available for operation at a much earlier stage in their disease Other authors have observed similar results, which are perhaps *a little less favourable only* because they were published earlier in a period of improving techniques in diagnosis and surgery, Tudor Edwards (1946), Ochsner and DeBakey (1948), Brock (1948), Churchill and his colleagues (1950)

TABLE

RESULTS OF 91 OPERATIONS FOR MALIGNANT DISEASE

Result	*Number of cases*	*Percentage*
Post-operative deaths	12	13
Died of recurrence in the 1st year	29	32
Died of recurrence in the 2nd year	11	12
Died of recurrence in the 3rd year	2	2
Deaths from other causes	6	7
Survivors	31	34

When the growth has extended beyond the lung either by glandular metastases or direct spread the prognosis after surgery is vastly altered. An analysis of the writer's cases shows that when there is no extension beyond the lung the 3 year survival rate was 60 per cent but when extension has occurred the survival rate is reduced to 15.7 per cent. A 60 per cent 3 year survival rate after resection for malignant disease offers a fair prognosis to patients and should be a great incentive to early diagnosis.

Deep x ray therapy

Radiotherapy has something to offer to patients with new growths of the lung but its possibilities are limited and it is employed mainly as a palliative. The [illegible] growths which occur in young people and rapidly produce symptoms of mediastinal obstruction. The prognosis in such cases is almost hopeless but the symptom [illegible] [illegible] antibiotic drugs should be given to clear up the infection before the treatment is started.

The best prospect for radiotherapy is probably in those cases of peripheral growth in which surgery is contra indicated only by old age or dyspnoea.

Palliative treatment

Apart from radiotherapy the palliative treatment of carcinoma of the lung consists of the relief of pain the relief of cough the treatment of infection and the relief of dyspnoea.

There is much that can be done to increase the comfort of patients with inoperable cancer of the lung. Morphine and its derivatives are invaluable and should be [illegible]

[illegible] superior sulcus tumours intercostal neurectomy is impractical as the nerve roots themselves are often involved. In

these cases the operation of cordotomy is often justifiable and earns the deep gratitude of the patient

Severe toxaemia and copious sputum may result from infection of the lung distal to the growth Remarkable symptomatic improvement can be obtained in such cases by the employment of suitable antibiotic drugs, of which penicillin is at present the one in most common use

Pleural effusion may cause distressing dyspnoea, and should be aspirated when indicated to relieve symptoms If the effusion is infected it should also be sterilized by the introduction of antibiotic drugs

ALVEOLAR-CELL CARCINOMA AND ADENOMATOSIS

This section would be incomplete without reference to a type of tumour which is receiving increasing attention in the literature, to which the reader is referred for further information

The condition is described as *alveolar-cell tumour, alveolar carcinoma* and *pulmonary adenomatosis* It bears a close similarity to a condition found in [illegible] South Africa [illegible] The main characteristics are [illegible]

acceptance of the nature and pathology of the lesion There is for instance no agreement that the alveoli normally have an epithelial lining Many pathologists regard the tumours as adenocarcinomas, which are showing bronchogenic dissemination Clinically the lesion behaves as a low grade malignant growth, causing haemoptysis, dyspnoea and cough Ultimately unless resection is successful, the patient dies of asphyxia, pneumonia or metastases As far as treatment is concerned surgical removal of the tumour offers the best hope of survival Ikeda (1945) has discussed this condition fully, and Griffith, McDonald and Clagett (1950) have reported 7 cases and made a statistical study of 51 other cases reported in the literature

SARCOMA OF THE LUNG

Primary sarcoma of the lung must be considered rare It is usually difficult to make the diagnosis during life without operation as these growths are seldom visible on bronchoscopy

Round-cell or spindle-cell growths may occur as highly malignant tumours in young people They may present with symptoms of pain or toxaemia and radiography will demonstrate a pulmonary mass In the absence of metastases they are best treated by a combination of surgery and deep x-ray therapy but the prognosis is poor

Fibro-sarcoma of the bronchus is a great rarity that may be detected on bronchoscopy, and may present with the signs and symptoms of bronchial obstruction These tumours are of low-grade malignancy and the prognosis after removal of the growth is good The subject is fully discussed by Black (1950) and by Carswell and Kraeft (1950) The writer has seen only 1 case of this condition, a child aged 2½ years who has survived for 7 years without evidence of recurrence after local excision of the tumour

Lympho-sarcoma usually involves the lung secondarily from an origin in the

mediastinum but there have been a few reported instances of lympho-sarcoma starting in the lung and presenting as an intrapulmonary mass The diagnosis in these cases is only made after resection which appears to be a successful procedure It should be combined with deep x ray therapy Maier (1948) describes a case of this nature

SECONDARY MALIGNANT TUMOURS OF THE LUNG

The lung is a common site for metastasis from malignant tumours in other parts of the body When metastasis occurs in the lung it is usually generalized and the evidence of the pulmonary lesion is masked by the other signs of dissemination of the growth

The secondary deposits in the lungs are caused by vascular embolic spread and consequently the lesions are usually multiple The radiological appearance is quite characteristic and consists of multiple rounded shadows throughout both lungs (Fig 156) As time passes the shadows steadily increase in size It is remarkable how symptomless pulmonary metastases may be often they cause no symptoms until they produce dyspnoea by their very bulk and the effects of pulmonary destruction Pain and haemoptysis are both unusual

Solitary metastases in the lung are uncommon and may cause difficulty in diagnosis they present as single rounded tumours in the radiograph and it is not always possible to differentiate them from a fresh primary peripheral lung tumour (Fig 157) Bronchoscopy sometimes reveals the true nature of the growth When there is no evidence of metastases elsewhere it is justifiable in some cases to resect the pulmonary lesion Seiler Clagett and McDonald (1950) have reviewed the literature on this subject and reported the results of pulmonary resection for metastatic malignant lesions at the Mayo clinic

ADENOMA OF THE BRONCHUS

Except for carcinoma bronchial adenoma is far the commonest and most important of the primary pulmonary neoplasms Although uncommon in comparison with carcinoma individual chest surgeons are accustomed to treat 2 or 3 fresh cases of adenoma every year Its inc dence can only be defined in relation to carcinoma In the writer s experience adenoma represents 10 per cent of operable pulmonary tumours Churchill and his colleagues (1950) record 30 pulmonary resections for adenoma in the period 1930–50 in relation to 171 resections for carcinoma It should be remembered that only about 10 per cent of carcinomas come to resection so it may be inferred that adenomas comprise only about 1 2 per cent of primary neoplasms of the lung

In contrast to carcinoma adenoma is commoner in women than men and occurs as a rule in a younger age group though it is occasionally observed even in men over 60 years of age

Pathology

Kramer (1930) first drew attention to the clinical importance of adenoma of the bronchus although pathological observations had been published earlier He suggested that the tumour arose in the mucous glands of the bronchus that it

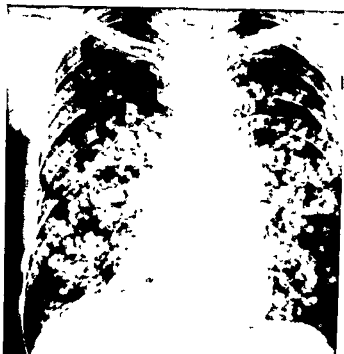


FIG. 156 —Multiple metastases in both lungs from carcinoma of the prostate



FIG 157 —A "cannon ball" secondary sarcoma Similar appearance is often produced by secondary hypernephroma

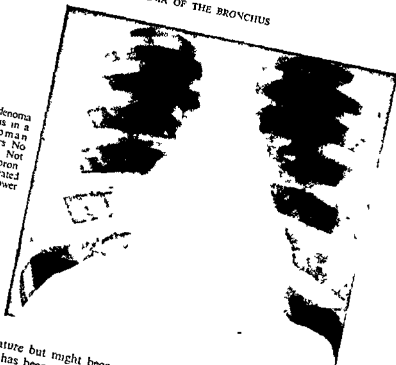


FIG 158 —Adenoma of bronchus in a young woman aged 21 years No symptoms Not visible on bronchoscopy Treated by right lower lobectomy

was of a benign nature but might become malignant occasionally During the past 20 years there has been a vast amount of discussion on the subject on the origin of the tumour and on its potential malignancy, but little real advance has been made on the views originally expressed by Kramer Womack and Graham (1938) and Graham and Womack (1945) have held that bronchial adenomas arise from a foetal bronchial bud that they have a dangerous tendency to malignancy and should be removed by total pneumonectomy

Much confusion has been caused by the divergent views of pathologists who have reported adenomas as carcinomas and *vice versa* Clinically there is no doubt that there is a group of cases of innocent epithelial tumours of the bronchus that run a benign course and these tumours can be recognized by experienced pathologists That there should be occasional examples of a malignant change is not a matter for surprise or acceptance of universal malignancy Foster Carter (1941) has pointed to the similarity between these lesions and the tumours of the parotid gland It is of the greatest importance to the patient that bronchial adenoma should not be confused with bronchogenic carcinoma for the prognosis of the latter is on the whole poor whereas the prognosis in adenoma is excellent provided correct diagnosis and treatment are applied

The tumours may occur in the trachea, but they arise as a rule in a major bronchus at or near a primary division They are thus usually visible on bronchoscopy, occasionally they arise peripherally, and are beyond bronchoscopic access (Fig 158) They may be entirely endobronchial but more commonly there is also an



FIG 159—Adenoma of bronchus showing distension of the bronchus by the tumour distal collapse, fibrosis and bronchiectasis Operation specimen



FIG 160—Bronchial adenoma (×250 Reduced one quarter on reproduction)

extra bronchial extension The tumour is polypoid and is covered by intact mucous membrane As it grows it distends the bronchus and compresses the surrounding tissues (Fig 159) Histologically the tumour is covered on its exposed surface by bronchial epithelium from which it is separated by a layer of connective tissue The cells of which the tumour is composed are arranged in solid acini, *they have a remarkably orderly and uniform appearance Mitotic figures are* rare (Fig 160)

Clinical course

As the tumour grows it obstructs the bronchus, and like certain forms of carcinoma it gives rise to bronchial obstruction, with complications due to bronchiectasis and infection (Fig 161) Before complete obstruction occurs it may give rise to symptoms such as cough, haemoptysis and dyspnoea Once infection has supervened the sputum becomes purulent and signs of toxaemia such as fever, malaise and loss of weight occur

The correct diagnosis can only be made by biopsy, otherwise the signs and symptoms closely resemble those of carcinoma Adenoma may be suspected as opposed to carcinoma by the length of history, which may extend over many years, and by the youth and sex of the patient Haemoptysis is a commoner symptom *in adenoma than in carcinoma, at times it may be severe and it may* occur as the only symptom with long intervals of freedom, or it may be associated with attacks of so called " pneumonia "



Fig 161 — Bronchial adenoma causing collapse of the right lower and middle lobes with secondary infection and infective changes in the upper lobe

Treatment

Before pulmonary resection became a relatively safe operation bronchial adenoma was treated by bronchoscopic methods by diathermic fulguration and radon implantation These methods although satisfactory in a few early cases failed to eradicate the extra bronchial portion of the growth and failed to deal with the suppurative complications distal to the growth They have been superseded as a general rule by open operation At thoracotomy it is possible to define the exact extent of the tumour If it is entirely endobronchial the bronchus may be opened and a complete local excision of the growth may be carried out the bronchus is sutured and no loss of pulmonary tissue occurs More commonly the lung distal to the tumour is irreparably damaged by obstruction and infection and it is necessary to perform a lobectomy and even in some cases a total pneumonectomy

HAMARTOMA OF THE LUNG

Chondromatous hamartoma

The term hamartoma was coined by Albrecht (1904) to apply to certain tumour like malformations in which there is an abnormal mixing or development of the normal components of an organ Although the term was not originally used by Albrecht for lesions of the lung it seems an apt word that has become generally accepted for certain benign tumours of the lung The incidence of this type of benign lesion is second only to bronchial adenoma

The commonest pulmonary hamartoma is a tumour like mass that contains



FIG 162 —Chondromatous hamartoma The glistening white chondroma is enveloped in a well defined capsule



FIG 163 —Chondromatous hamartoma showing cartilage fibrous tissue and clefts lined by epithelium (× 44 Reduced one quarter on reproduction)

elements of normal pulmonary tissue The mass is likely to contain cartilage, epithelium, fibrous tissue fat and vascular elements They are liable to be described as tumours of their most dominant tissue component The most common and characteristic hamartoma is mainly cartilaginous and may be reported as a chondroma (Fig 162) A careful histological examination reveals that in addition to cartilage there are clefts lined by columnar or cuboidal epithelium and separate areas of fibrous and fatty tissue (Fig 163)

The rate of growth of these lesions has not been determined , it may be that growth occurs only *pari passu* with that of the individual There is no evidence of malignant change, but such a possibility should not be disregarded Carlsen and Kiaer (1950) have described the pathology of these tumours in considerable detail

Cartilage is not necessarily the main constituent, and in some instances the tumour may be described as a fibroma a fibro adenoma or even a lipoma Many of the reported examples of this condition have been chance findings at autopsy or in operation specimens McDonald, Harrington and Clagett (1945) reported 23 cases, of which 20 were found at necropsy and 3 were removed surgically Most of these tumours " occur in peripheral parts of the lung, they cause no symptoms and require no treatment (Fig 164) When the lesion is endobronchial it causes bronchial obstruction with complications of distal collapse and infection If the lesion is accessible to the bronchoscope the diagnosis may be proved by biopsy In these cases, in which the lesion produces symptoms, the correct treatment is

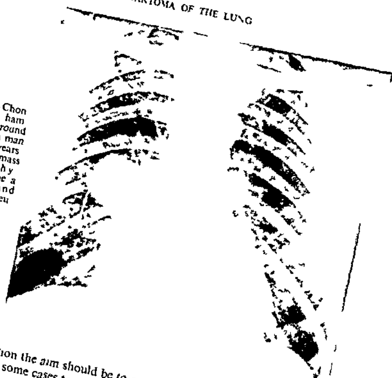


FIG 164 — Chondromatous hamartoma A round shadow in a man aged 50 years found on mass radiography Thought to be a carcinoma and treated by pneumonectomy

excision At operation the aim should be to conserve all healthy lung tissue thus it may be possible in some cases to excise the tumour by bronchotomy in others a limited lung resection is necessary

Vascular hamartoma

The vascular hamartomas constitute quite a separate group of developmental errors or aberrations In recent years there has been a great increase in the literature on this subject and it seems clear that vascular lesions of the lung are more common than was believed formerly A number of different terms have been employed in the nomenclature The lesions may range from small telangiectases scattered throughout both lungs to a solitary pulsating vascular mass composed of a tortuous dilated branch of the pulmonary artery which communicates directly with a pulmonary vein It is because of the wide range in the anatomical variety of these lesions that terms such as pulmonary telangiectasis cavernous haemangioma arteriovenous aneurysm and arteriovenous fistula have been employed

The multiple lesions may be associated with evidence of telangiectases elsewhere particularly on the face the lips and the buccal mucous membrane The writer has observed one example in a patient with hereditary familial telangiectasis Goldman (1947) has reported the condition in two brothers Haemoptysis is not necessarily a common symptom though it does occur in some cases and has been recorded as a fatal complication



(a)

FIG 165 —(*a* and *b*) Vascular hamartoma (multiple cavernous haemangiomas) Indefinite rounded shadows in both lower lobes in a woman who showed the classical signs of cyanosis polycythaemia and clubbing with telangiectasis on the face and lips



(b)



Fig 166—Vascular hamartoma (solitary arteriovenous fistula) in a young man with dyspnoea cyanosis and clubbing Treated by right lower lobectomy

The signs and symptoms depend in their severity on the degree of shunt of deoxygenated blood from the pulmonary artery to the pulmonary vein Maier and his associates (1948) record a case in which 58 per cent of the total cardiac output at rest passed through the abnormal arteriovenous communication According to the proportion of blood that fails to become oxygenated in the lungs there are signs of cyanosis polycythaemia and clubbing of the fingers As a result of anoxia the patients are breathless and many suffer from attacks of faintness These signs may suggest a diagnosis of congenital heart disease such as the tetralogy of Fallot and they have in the past no doubt led to an erroneous assumption that the patient suffers from polycythaemia vera The key to correct diagnosis lies in a careful examination of the lungs Auscultation over the lesion will often disclose a continuous murmur that becomes louder on inspiration Radiology will reveal in the lung field a shadow or shadows which increase in size on inspiration (Fig 165) Kymography and screening will show expansile pulsation and tomography frequently demonstrates the continuity between the lesion and the vascular shadows of the pulmonary artery and veins Angiography with a contrast medium is a refinement of diagnosis which is seldom if ever required

The only treatment possible for these lesions is excision The need for treatment depends on the severity of the symptoms If the arteriovenous shunt in the pulmonary circulation is small there may be no disability and there is no urgent need for operation If the lesions are multiple and bilateral total excision is impossible considerable symptomatic improvement can be attained by excision

of the major lesions in which a shunt occurs In the case of a large solitary arteriovenous fistula a cure can be expected by lobectomy, and this is the operation of choice (Fig 166) In some instances such as that reported by Jones and Thompson (1944) total pneumonectomy is required

Local excision of the lesions, even when large, is possible if temporary clamps are applied to the pulmonary artery and veins, but there is a risk of cerebral embolism from a clot in the proximal part of the ligated vein This accident has occurred in the experience of the writer Janes (1944) was the first to report a case treated successfully by bilateral local excision

NEUROGENIC TUMOURS

Tumours may arise from the nerves that supply the bronchi They must be considered extremely rare The writer has observed 2 cases in which a neurofibroma arose in close connexion with a bronchus Both were removed without the necessity of resecting lung tissue Diveley and Daniel (1951) report a case in which a neurofibroma was situated in the middle lobe and treated by lobectomy.

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CHAPTER 26

BORNHOLM DISEASE

KENNETH M A PERRY

THIS epidemic disease, variously known as epidemic pleurodynia, epidemic myalgia, or "The Devil's Grip" is characterized by the sudden occurrence of violent pain in the lower part of the thorax which tends to spread to the abdomen The incubation period is 2–5 days and patients remain infective as long as the pain persists The disease was first described in Norway in 1872 by Daee, while two years later Finsen reported that outbreaks had occurred in Iceland in 1856 and 1863 It attracted little attention until Sylvest in 1930 described 93 cases in patients between 5 months and 59 years of age which occurred on the island of Bornholm in the Baltic Since this time reports of many outbreaks have been reported, both in Great Britain and in the United States of America The disease usually occurs in warm weather, when there is a considerable period of continuously high humidity It occurs mostly in young adults and rather more commonly in females than males The onset is abrupt with pain in the epigastrium and hypochondriac region, which spreads into one side of the chest and is accentuated by breathing Ronald (1942) noted that in addition to the abdominal muscles, there was particular involvement of the muscles around the shoulder girdle and neck The affected muscles are tender and swollen, and the disease is accompanied by fever usually up to 101° F , but sometimes higher, while frontal headache occurs in more than half the cases, and occasionally vomiting, diarrhoea, and epistaxis Paraesthesia in the arms and hands occurs especially in females There are rarely any signs in the chest, and the spleen is not enlarged Jamieson and Prinsley (1947) and Gray and Carter (1948) record cases in which a pleural friction rub was heard, and the latter authors recorded a case in which there was palpable fremitus In a quarter of the cases one or more relapses takes place The tenderness and swelling of the muscles and pain on exertion may last several weeks or months after the attack has subsided Complications such as pleurisy in rare cases with effusion, conjunctivitis, orchitis, pericarditis, pneumonia and otitis media have been reported but mostly the disease follows a benign course and results in complete recovery Nichamin (1945) noted a marked reduction in haemoglobin level, while the red cells numbered less than four millions The white cell counts seem to vary widely, with no particular abnormality in the differential counts The sedimentation rate is usually raised Screening the chest reveals a freely moving diaphragm, and skiagrams show no abnormalities in the lung fields The disease is of considerable importance because of the differential diagnosis from pleurisy

The aetiological organism has not yet been identified but the Annual Report of the U S Public Health Service for 1944, records the fact that the blood, spinal fluid and nasal washings of the human cases of the disease have yielded an agent pathogenic to monkeys Findlay and Howard (1950) carried the matter a stage

further by showing that sera from patients fixed complement with Coxsackie virus 2 antigen

Patients should be treated by being kept in bed until 48 hours after the pyrexia and pain have subsided There is no specific treatment but ordinary methods should be used for the relief of pain

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CHAPTER 27

TUMOURS OF THE CHEST WALL

GEOFFREY FLAVELL

TUMOURS arising from the chest wall may be divided into those that are superficial and those that are deep. The former, comprising mainly innocent lesions such as cysts, naevi and lipomas, originate in the skin and subcutaneous tissues and are therefore not fixed; while the latter tend to involve the ribs and intercostal muscles, are commonly fixed, and must all be regarded either as malignant or potentially so. Neither group differs materially from those growths to be found elsewhere on the body surface, or in skeletal structures, and those classed as superficial do not warrant further description; but the deep and fixed variety pose, from their situation, particular diagnostic and surgical problems. In a recent review of 48 such tumours, Blades and Paul (1950) found 28 benign (of which at least 7 were potentially malignant), and 20 (47 per cent) malignant; while it has been estimated that primary malignant tumours of the ribs and sternum comprise some 10 per cent of all primary malignant bone tumours.

The symptoms caused by chest wall growths are principally pain and swelling. Pain may long precede the latter, since the tumour may grow into the pleural cavity rather than present upon the surface; and it is usually boring in nature, sometimes pleuritic, or may be referred along the segmental nerves to produce abdominal pain, leading to a mistaken diagnosis of gall bladder or other intra-abdominal disease. Blades and Paul found an average of 6 months elapsed between the onset of such pain and x-ray investigation of the affected part, 2 months of which was spent in the treatment of "neuritis". When pain is constant and severe, malignancy is to be feared; on the other hand tumour masses may be quite painless and attain great size. The frequency with which these lesions are associated by patients with previous trauma is well-known, but it is doubtful if a true relationship exists; although pain (or swelling) persisting in a rib for any considerable period after injury should be most carefully investigated. Idiopathic fractures occasionally occur in the ribs, most commonly in the first, second, seventh and eighth. These have been attributed to stresses resulting from coughing and called "cough fractures" (Annotation 1946). Their importance lies in their superficial resemblance to a spontaneous fracture through a tumour of rib with which they must not be confused.

In the past many "endotheliomas" of the pleura, usually accompanied by blood-stained pleural effusions, have been described. There is now little doubt that the great majority of these were in fact undifferentiated bronchial carcinomas which invaded the pleura at an early stage and spread rapidly upon its surface; and that true endotheliomas (or mesotheliomas) arising from the pleura itself are exceptionally rare, if indeed they occur at all. The soft tissues of the th... rise to fibromas, lipomas, or fibrosarcomas wh... to the pleura, or involve it, but differ in no way

neuromas or neurilemmomas arise from intercostal nerves expand the intercostal spaces and cause smooth pressure erosion of related ribs They may be isolated or part of generalized von Recklinghausen s disease while those much more common situated in the paravertebral gutter, are classed among the tumours of the mediastinum It remains therefore to consider those growths which principally arise in the bony structures of the thorax

Secondary growths

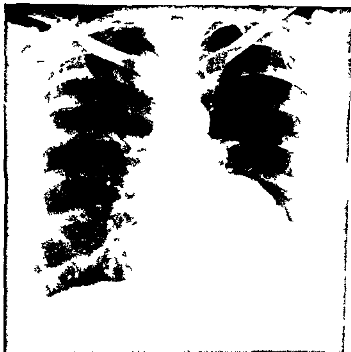
Metastases especially from carcinoma of the thyroid kidney and breast are the commonest of chest wall tumours comprising about 38 per cent of them They may be more conspicuous than their primary source and difficult to distinguish radiologically from intrinsic tumours of rib When very vascular they sometimes present as pulsating masses to be differentiated from aortic aneurysm which likewise may produce a thoracic swelling which is not always pulsatile usually on the right of the sternum Growths of the mediastinum in particular dermoids have on occasion a pre sternal element linked by a dumb-bell extension to the mass within and carcinoma of the lung often invades the chest wall especially in the superior sulcus where it produces the painful Pancoast syndrome

Inflammatory lesions

Specific and inflammatory lesions to be distinguished are tuberculous or pyogenic osteomyelitis empyema necessitatis or more rarely typhoid osteochondritis or gummas Tuberculous lymphatic glands situated at the anterior ends of intercostal spaces often break down into a tense and fixed small cold abscess which is none the less fluctuant and from which sterile pus can be aspirated Other disorders that occasionally simulate growths are mal united fractures congenital or rachitic deformities or marked unilateral protusion of the bony cage due to scoliosis A non specific non suppurative condition causing painful swelling usually of one costo-chondral junction is Tietze s disease Seldom encountered in civil practice it is remarkably frequent among young military recruits and often leads to unnecessary intervention The wearing of packs has been dubiously blamed for its appearance It runs a long course the swelling altering in size but never breaking down while histologically some fibrotic change is found in the cartilage with periarticular swelling of the soft tissues

Primary tumours of the ribs and sternum

Most tumours of the ribs and sternum grow from cartilage and can be classed either as chondromas or chondrosarcomas These growths comprised 38 per cent of the series of 81 collected by Sommer and Major in 1942 and 65 per cent of Dorner and Marcy s 23 examples in 1948 The ribs are the site in about 80 per cent of neoplasms the sternum 20 per cent while of cartilaginous tumours some 68 per cent are to be found situated near the costo-chondral junction the manubrium being the most commonly affected portion of the sternum A good deal of confusion in nomenclature exists such terms as osteochondroma osteochondrosarcoma and chondromyxosarcoma tending to disguise the essentially cartilaginous character of the growths in describing bone formation or mucoid degeneration both of which are merely secondary features Osteogenic sarcoma predominating



F G 167 (*a* and *b*) A la ge chondroma n ol ng the pos ter or end of the left 10 h r b

(*a*)



(*b*)

over chondrosarcoma elsewhere is conspicuously rare in the chest as is also the osteoclastoma a total of only 9 instances being recorded in literature

Cartilaginous tumours are hard and nodular with occasional softer or even fluctuant areas If the history is long as it frequently is they may attain considerable size X ray examination is seldom conclusive showing merely destroyed bone or an expanded and thinned out cortex though irregular flecks of calcification are common and typical (Fig 167) A firm distinction between the innocent and the malignant can in many cases be made only histologically Indeed there is a dangerous tendency to underrate the malignant potentialities of apparently innocent chondromas for mitoses are exceptional and the presence of atypical nuclei or giant cartilage cells must be recognized as indicating malignancy Similarly some portions of a tumour may seem innocent while others are clearly invasive Enchondral bone formation suggests low malignancy while the presence of mitoses implies high malignancy Most tumours are encapsulated to some extent and even malignant ones can often be shelled out with deceptive ease The advancing edge of the growth remains discrete first displacing and distorting adjacent soft tissues then invading them Local recurrence often repeated, follows inadequate removal and may show increased malignancy Metastases are unusual appearing only late in the disease and being generally blood borne Chondrosarcoma is known to develop from innocent chondroma and since a pre operative diagnosis cannot with certainty be made all should be radically extirpated along with adjoining muscle pleura or any related structures Defects resulting in the chest wall permit paradoxical respiration and must therefore be repaired either by interposing rib grafts from above or below by rigid prostheses or, if small by transposing periosteum across them so that new bone is formed X ray therapy is useless in these as in most other chest wall tumours with the exception of Ewing s sarcoma and those arising from lymphoid tissue

The next common cause of rib tumour is the fibrous dysplasia described by Schlumberger Symptoms may be altogether lacking but a history of trauma is often given as the cause of the swelling The rib is moderately expanded x ray examination revealing a fusiform area with semi translucent centre surrounded by the narrowed cortex (Fig 168) Histologically the medulla is replaced by masses of young fibrous tissue in whorls and trabeculae with patches of bony metaplasia Malignant change does not occur and providing always that a positive diagnosis has been made local excision of the affected rib suffices

Plasmocytomas and single myelomas of rib are mentioned in most series and may be more common than recorded numbers suggest (Fig 169) The latter have the character of myelomas elsewhere in the body including a raised plasma protein though Bence Jones protein is not usually present in the urine until late in the disease A similar tumour accompanies eosinophilic granuloma and may like myeloma be solitary or multiple involving in the latter case all the bones except the hands and feet Male children and adolescents are chiefly affected the symptoms of local tenderness and sometimes pathological fracture being of short duration Leucocytosis with eosinophilia pyrexia and loss of weight lead to the diagnosis and the prognosis following excision is good The condition is believed by some to manifest the same basic disorder as Letterer Siwe or Hand Schuller Christian

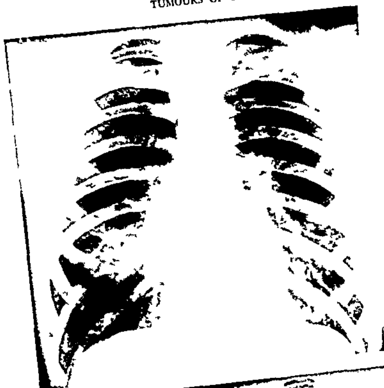


Fig 168—Fibrous dysplasia involving the posterior portion of the right 9th rib

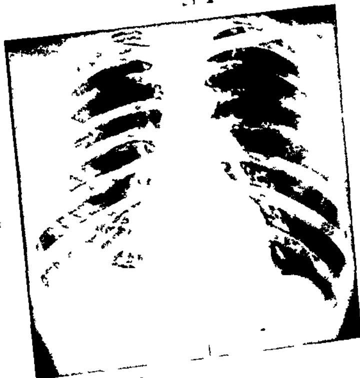


Fig 169—A solitary myeloma in the mid portion of the right 10th rib

disease Osteitis fibrosa, which is sometimes caused by a parathyroid tumour when the blood calcium will be raised, will on occasion cause one or more tumours in the bony thoracic cage

Ewing's tumour in ribs is rare, only 18 cases having been reported up to the end of 1942, but when occurring it is found usually in patients during the second decade of life, and should be treated by deep x ray therapy

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CHAPTER 28

DISEASES OF THE MEDIASTINUM

KENNETH M A PERRY

THE MEDIASTINUM occupies the middle of the chest and lies between the two lungs In it lie the trachea, oesophagus and heart Diseases of the oesophagus and heart are usually considered in treatises on the digestive tract or cardiology and will not be discussed in this book Enlargement of mediastinal glands results from reticulosis, sarcoidosis and secondary carcinoma and is discussed in the chapter dealing with these subjects

MEDIASTINAL DISPLACEMENTS

In a healthy patient the mediastinum is freely mobile and is readily displaced by disease in either lung Even lying on one side causes the mediastinum to shift to the dependent position The mediastinum shifts towards the side of the lesion when there is fibrosis in the lung or when collapse occurs, and it is pushed away from the side of the lesion when air or fluid is present in the pleural space In certain cases of pneumothorax there may be ballooning of the pleural cavity between the aorta and the oesophagus giving rise to a mediastinal hernia (Fig 170) Displacement by fibrosis occurs particularly in unilateral upper lobe fibroid phthisis, while collapse giving rise to bronchiectasis or caused by neoplasm is also a common cause of shift of the mediastinum Perhaps the most common cause, however, is a pleural effusion and if this is present without shift it may be suspected that there is an underlying carcinoma fixing the mediastinum Expanding tumours and aneurysms of the aorta are other conditions which may cause the mediastinum to be displaced In kypho scoliosis there is gross distortion and twisting of the mediastinum which is nearly always associated with severe chronic bronchitis and emphysema

Mediastinal displacement gives rise to no symptoms, unless it occurs suddenly, when the patient may complain of sudden collapse and weakness On examination the trachea will be felt to be displaced and the apex beat and area of cardiac dullness will also be displaced Fluoroscopy is sometimes of great value since it may show a swinging shift of the mediastinum during respiration, which is not shown by the straight skiagram Normally, however, mediastinal shift is quite evident in a skiagram of the chest Mediastinal displacement in itself does not require treatment This should always be directed to the underlying condition which has given rise to the shift

MEDIASTINAL EMPHYSEMA

Air may reach the mediastinum along the fascial planes of the neck, through a perforation of the trachea, oesophagus or bronchus, from the retro peritoneal space, or from the interstitial tissues of the lung, and it is theoretically possible

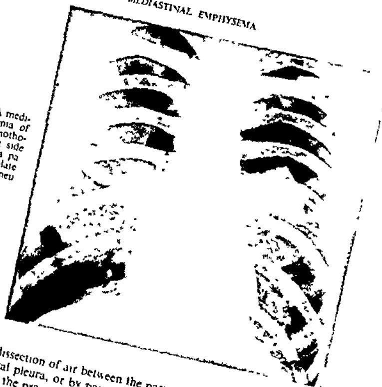


FIG 170.—A mediastinal hernia of left pneumothorax to right side of chest in a patient with bilateral artificial pneumothoraces

for it to happen by dissection of air between the parietal pleura and the chest wall or between the visceral pleura, or by passage of air from the pleural cavity to the mediastinum From the practical point of view the ways that need consideration are, spread along the fascial planes of the neck, and interstitial pulmonary emphysema It is well known that the former may occur, and mediastinal emphysema and bilateral spontaneous pneumothorax are sometimes complications of operations on the neck, particularly thyroidectomy and tracheotomy, and more rarely tonsillectomy and other operations Since, however, these operations are always accompanied by anaesthesia through intratracheal tubes, and tracheal and bronchial obstruction and frequently coughing are all familiar causes of interstitial emphysema of the lung, it seems probable that these complications often result from rupture of pulmonary alveoli rather than direct aspiration of air into the mediastinum The causes of interstitial pulmonary emphysema are first, traumatic, either as a result of injury to the chest, with or without fracture of ribs, or from operations of the chest including the induction of an artificial pneumothorax Secondly, increase of pulmonary pressure caused by straining with a closed glottis, as may occur in lifting heavy weights, straining at a stool, during child birth (Fig 171) or during attempts to resuscitate the new born, or as a result of partial or complete occlusion of the trachea or bronchus accompanied by coughing, as may occur during anaesthesia, in asthmatic attacks, in chronic bronchitis, from inhaled foreign bodies, or during the course of whooping cough or pneumonia Thirdly, it may result from the spontaneous rupture of the alveoli This latter condition is of

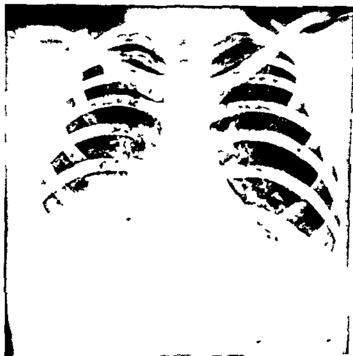




FIG 171 — (a) Mediastinal emphysema which developed in a woman during labour (b) lateral view showing the emphysema under the sternum

particular interest in that it may occur in an apparently healthy individual without any evidence of increased intra alveolar pressure Since the condition has a tendency to recur it seems possible that the cause is an inherited defect in the tissue of the alveolar wall

Mediastinal emphysema may remain confined within the mediastinum or it may escape into the subcutaneous and deep tissues of the neck through the dia phragmatic openings into the retro peritoneal tissue and into the pleural cavity A small amount of air in the mediastinum will probably cause only trivial symptoms Larger amounts will cause pain in the chest and difficulty in breathing and the patient may lie flat in bed trying to suppress his cough Further increase in the mediastinal pressure gives rise to symptoms similar to those of increased pericardial pressure the veins collapse and the flow of blood into the heart is diminished If the pressure rises sufficiently high the circulation may come to a standstill as this happens shortness of breath cyanosis and pulmonary oedema may develop

On physical examination of the patient if there is subcutaneous emphysema of the neck it will be evident to the touch The most characteristic physical sign of mediastinal emphysema however consists of crackles bubbles or churning sounds heard with each contraction of the heart These may be heard only during systole but sometimes they occur throughout the whole cardiac cycle with accentuation during systole Besides these characteristic signs there may be obliteration of the cardiac dullness and perhaps evidence of increased mediastinal pressure presenting as dyspnoea cyanosis engorged veins and circulatory failure If pneumothoraces are present the signs of that condition will be evident Skia grams of the chest may show collections of air quite plainly but sometimes only a few streaks may be seen in the mediastinal tissue between the heart and the chest wall in the lateral view A sharply defined translucent band outlining the peri- cardium especially along the upper part of the left border is a characteristic finding Pneumothoraces are evident if they are present

Treatment

A small amount of air in the mediastinum is of no consequence as the air is soon absorbed If tension pneumothoraces are present it is important to insert a needle into the pleural cavity and attach it to an under water drain If pressure on the mediastinum itself gets high a catheter inserted into it through the jugulum may give relief If there is cardiac embarrassment this may be treated with oxygen and stimulants given to combat anoxia and shock Morphine should be given to relieve pain

ACUTE MEDIASTINITIS

Acute mediastinitis may occur with or without suppuration Simple mediastinitis mostly results from lacerations of the oesophagus and trachea or from injuries of the chest wall or sternum which cause haemorrhage It may however spread from inflammatory processes *in the lungs pleurae pericardium peritoneum* sternum or vertebrae Suppurative mediastinitis is a more common condition and Neuhof and Jemerin (1943) and Pearse (1938) have shown that about half the cases result from trauma particularly perforation of the oesophagus This may occur spontaneously in cases of carcinoma of the oesophagus but more often it is

the result of the passage of instruments, oesophagoscopes, gastroscopes or stomach tubes It may also result from impacted foreign bodies especially rabbit or chicken bones A further 25 per cent of cases are the result of spreading infection from the neck, acute pharyngitis, retropharyngeal abscess, quinsies and suppurative cervical adenitis Other causes include the spread from adjacent organs, thus pneumonia, bronchiectasis and lung abscess may cause acute mediastinitis ; as may periostitis and osteomyelitis of the vertebrae, sternum or ribs, and suppuration in the mediastinal glands Spread may occur from pleura, pericardium or peritoneum Suppuration in a hydatid or dermoid cyst may break into the mediastinum Lastly, the infection may be blood borne in cases of bacterial endocarditis, pyaemia, erysipelas or typhoid fever

Pathology

In simple mediastinitis enlargement of the lymph nodes with hyperaemia of mediastinal connective tissues and inflammatory oedema are the only pathological changes When suppuration occurs it may be limited to one anatomical subdivision of the mediastium or may spread from one part to another It may spread up into the neck, down to the abdomen or point through the chest wall Abscesses have also been known to rupture into the oesophagus, trachea, aorta, pleura or pericardium

Symptoms

These vary according to the cause and the virulence of the infection ; there are those symptoms caused by infection and those caused by pressure Following perforation of the oesophagus symptoms will appear immediately ; in other causes the onset may be more insidious There is severe pain under the sternum radiating to the throat, back and shoulders associated with dysphagia, dyspnoea, fever and sometimes rigors There may be cough with purulent sputum, and the cough may be brassy as a result of pressure on the trachea Other pressure symptoms include hoarseness from involvement of the left recurrent laryngeal nerve, neuralgic pains from pressure on the intercostal nerves or brachial plexus, and superior mediastinal obstruction from pressure on the great veins

Physical examination will usually reveal little The patient will look ill and be cyanosed and dyspnoeic There may be stridor Enlargement of veins from superior mediastinal obstruction is more common in chronic mediastinitis There may be painful areas over the spine or sternum Subcutaneous emphysema may result from perforation of the oesophagus and torticollis from suppuration in the carotid sheath Local redness, oedema and fluctuation may be caused by the pointing of the abscess Dullness and distant breath sounds to one side of the sternum are sometimes found Skiagrams, particularly if they are over-penetrated, may reveal a broadening of the mediastinal shadow, the trachea may be pushed forward and a foreign body may be revealed ; on screening a superior mediastinal abscess may appear as a pulsating shadow Blood examination will always show a polymorphonuclear leucocytosis

Prognosis

The prognosis is always grave and the condition is always acute, rapidly progressive and frequently fatal The abscess may rupture into the oesophagus or

a bronchus and this is often providential drainage The suppuration, however, may spread to cause empyema, pericarditis or peritonitis Gangrene of the mediastinum, however, may result and is always fatal The prognosis is best when the disease is localized to the anterior mediastinum

Treatment

Foreign bodies should be immediately removed from the oesophagus and great care should be exercised in passing instruments when there is reason to think the oesophagus may be friable *In* cervical suppuration attempts may be made to block the retro visceral space by packing the fascial plane If mediastinal suppuration is already present attempts should be made to drain it as soon as possible, either by a posterior mediastinotomy or by resection of a costal cartilage or part of the sternum Penicillin, sulphonamides or the appropriate chemotherapeutic agent to which the causal organism is sensitive should always be administered If the oesophagus is perforated artificial feeding may be given through a Ryles tube, though it is usually more satisfactory to perform a gastrostomy

CHRONIC MEDIASTINITIS

Chronic fibrous mediastinitis may be the ultimate result of the acute suppurative condition in the mediastinum which has already been described The condition is most often caused by trauma, which has resulted in haemorrhage and organization, severe acute rheumatism that has caused a pericarditis, polyserositis, Pick's disease, tuberculosis, syphilis, actinomycosis, and Hodgkin's disease

Chronic mediastinitis frequently results in complete occlusion or thrombosis of the superior vena cava, an occurrence which was first described by Hallet in 1848 This condition, however, may also result from idiopathic thrombosis, thrombophlebitis, and pressure from mediastinal tumours, Hodgkin's disease, retrosternal thyroids and aneurysms Ochsner and Dixon (1936) reviewed the world literature and collected 120 cases of which 28 were due to chronic mediastinitis. Of these, the mediastinitis in 11 cases was caused by syphilis, in 10 by tuberculosis, in 1 by pyogenic infection, in 1 resulted from trauma and in the remaining 5 was of unknown aetiology Fischer (1904) first recorded actinomycosis as a cause and since then Cope (1938) has suggested that primary thoracic actinomycosis results from the fungus penetrating the wall of the oesophagus Mountsey (1947) has recently reviewed this subject and cases from other causes have been reported in recent years by Tubbs (1946) and Renbourn (1946)

Symptomatology

Symptoms mainly result from pressure or obstruction They include substernal pain, shortness of breath, hoarseness, dysphagia and Horner s syndrome, resulting from pressure on recurrent laryngeal and sympathetic nerves, trachea, bronchi, oesophagus and sternum Pottenger's sign is a deviation of the tongue to the affected side with fibrillar twitching of the muscle fibres It may be caused by a reflex connexion between the vagus and hypoglossal nerves

The principal symptoms, however, are attributed to superior mediastinal obstruction This causes swelling of the face, neck and arms, and on examination these will be found to be swollen and oedematous, while the subcutaneous veins on the

front of the chest and abdomen are dilated and the flow in these will be downwards Carlson (1934) states that obstruction of the superior vena cava is more severe when it occurs below the entrance of the azygos vein and Hinshaw and Rutledge (1942) have shown by infra red photography that enlarged veins extending over the costal margin on to the abdominal wall are seen only when the azygos is included in the obstruction

X ray examination may help in the diagnosis by showing widening of the superior mediastinal shadow, but this by itself is not sufficient to warrant a diagnosis of chronic mediastinitis in the absence of obstructive symptoms

Treatment

Venesection frequently gives temporary relief to patients with chronic medi astinitis Syphilitic cases should be treated with arsenic, bismuth and mercury as described in the chapter on syphilis Actinomycosis will respond to treatment with four million units of penicillin daily for 6 8 weeks

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superior vena cava , but Tubbs (1946) states that it is not warranted, as it has been shown to be hazardous and often, if not always fruitless In addition there are many cases reported who show that the condition is compatible with a long life without discomfort

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CHAPTER 29

CYSTS AND TUMOURS OF THE MEDIASTINUM

GEOFFREY FLAVELL

UNTIL comparatively recently the mediastinum was regarded almost as an abstract anatomical convention, and it is only in recent years that advances in radiology and in the techniques of thoracic surgery have revealed its practical importance and its pathological diversity In particular the increasing use of mass radiography has led to the early recognition of a bewildering variety of cysts and tumours, and to their early removal Differing in their anatomical relationship within the mediastinum and in their histological character, many of these lesions none the less owe their origin to a common factor—the developmental complexity of the region For it is upon this narrow stage that the more caudal of the branchial arches develop, that the primitive foregut buds off its pulmonary diverticulum, which in turn descends into the thorax branching and dividing into the ultimate elaboration of the bronchial tree, that the enteron itself differentiates that the heart and great vessels undergo their foetal mutations to reach their adult state that the pleural and pericardial cavities are fashioned from the infoldings of the simple coelom, and the whole cut off by the formation of the diaphragm from the septum transversum In addition to those growths which may be attributed to developmental faults, there remain others arising from the great diversity of adult tissues actually within or closely related to the mediastinum, and the richness of its lymphatic connexions in turn make it a frequent site for metastatic deposits from more distant organs, in particular from the lung Such secondary growths will not be considered here, though they inevitably feature in the differential diagnosis of primary tumours as they may occur where ever lymphoid tissue is to be found in the mediastinum

Investigation of mediastinal growths must always include good lateral, and if necessary, oblique skiagrams, as well as antero posterior views Radiological screening is indispensable for testing mobility and watching pulsation A barium swallow shows displacement or obstruction of the oesophagus, while tomography is sometimes useful in fixing exact relationship and revealing cavities Bronchoscopy and oesophagoscopy are chiefly of value in excluding other growths, but may fix a point of compression from without The induction of an artificial pneumothorax or pneumoperitoneum may assist diagnosis Thoracoscopy has been employed, but is seldom conclusive

Neuromas, enterogenous cysts, and meningeal growths will be found posteriorly, dermoids, teratomas, cysts of the pericardium, thymomas, and thyroid adenomas anteriorly But others occur in any situation and much else remains to be differentiated

Aneurysms of the great vessels, or of the heart, may be confused with new growths, and must be distinguished from them by radiological evidence of

pulsation, by demonstrating their continuity with the vascular structures, if necessary by angiography, by serological reactions, and characteristic physical signs The oesophagus, when dilated and full of food, as may happen in long-standing cardiospasm, adopts an S shaped position in the mediastinum, bulging to the right of its upper portion and to the left lower down In either situation it casts a rounded shadow which may greatly resemble a mediastinal tumour, but can readily be distinguished by a history of dysphagia and a barium swallow In similar fashion diaphragmatic herniation into, or beside the mediastinum can be recognized by barium followed through the gut Herniation of a lung across the midline often produces an appearance simulating a cyst wall, and scoliosis or vertebral disease distorts x ray appearances Radiography of the region in infants and young children is fruitful of equivocal "tumours", since the mediastinum is highly mobile at this age and it is difficult to obtain a strictly centred film Thus a little displacement or rotation of the heart and other structures casts disproportionate and unexpected shadows not always easy to interpret

All these possibilities as well as that of carcinoma of the lung or of the oesophagus, must be borne in mind when the diagnosis of an abnormal mediastinal shadow is to be attempted, and when each in turn has been eliminated there remains to differentiate the wide field of primary cysts and tumours Among these, characteristics of appearance and of position will sometimes permit a positive choice, and often an intelligent guess, as to their nature, but rarely indeed can there be certainty of diagnosis, and less frequently still can there be any true assurance as to a tumour s innocence or malignancy It is generally accepted that some 10 per cent of all innocent mediastinal cysts and tumours eventually undergo malignant degeneration, the proportion being higher among teratomas and some neurogenic growths, and lower in such groups as the simple cysts

Whether or not eventual malignancy is their fate, all are liable to enlarge, and from their situation to cause such symptoms as dyspnoea by pressure upon the adjacent bronchi or trachea, dysphagia by compressing the oesophagus, engorgement of neck veins and tachycardia by obstruction of the great vessels and displacement of the heart, and eventually all of these by a generalized blockage of the mediastinum

Cysts are prone to establish fistulous communications with the bronchi or with the pleural cavities, generally combined with secondary infection of their contents Such complications may carry fatal consequences Haemorrhage can occur into them Intrathoracic goitres are likely eventually to undergo toxic degeneration, tumours of the thymus to be associated with myasthenia gravis, enterogenous cysts may develop peptic ulceration and perforate, hydatid cysts disseminate their infestation It is thus evident, quite apart from diagnostic uncertainty, and from the omnipresent and substantial risk of malignant change, that no case exists for tolerating these tumours, and that all, unless the age or condition of the patient forbids, should be surgically removed

The relative frequency with which they variously occur is not easy to assess with accuracy since few surgeons trouble to record the commoner varieties, and classification of more unusual types remains unstandardized There is little

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(a)



(b)

FIG 172—(a) A typical neurofibroma Note the characteristic position in the paravertebral gutter (b) A right artificial pneumothorax has been induced but the tumour has not descended with the lung showing that it is extra pulmonary

however that neuromas top the list, followed by bronchogenic cysts d and teratomas, various tumours of lymphoid tissue, pericardial cysts r and enterogenous cysts, in that order, with rarer lesions following retrosternal thyroid enlargements are included they are probably st of all and certainly must be considered among growths of the superior

diastinal tumours removed in United States Army thoracic surgical period of 3 years, 90 were innocent, 15 malignant Among the 2) neuromas 23 bronchogenic cysts 14 dermoids and teratomas al cysts, the malignant numbered 6 teratomas, 4 cases of Hodg 2 lymphoblastomas, 2 thymomas, and a neurosarcoma Uhrich 60 primary mediastinal tumours removed at the Mayo Clinic s but roughly similar proportions

NEUROGENIC TUMOURS

te posterior mediastinum are, in 92 per cent of cases, neurogenic occurring elsewhere in the mediastinum are excessively rare, but wn They are divided broadly into those which originate from ths generally called neurofibromas or neurilemmomas, and those cell predominate, and which arise from the sympathetic ganglia n 1944 Kent collected 78 cases from the literature and found per cent) malignant He added 18, classifying 41 per cent int Harrington in 1949 recorded 51 neuromas of which 5 9 per cent malignant Godwin and associates in 1950 described 24 cases, 2 (8 per cent)

s and ganglioneuromas characteristically occupy a position r, evidently closely related to the sympathetic chain er in the upper part of the chest than in the lower but e in the gutter The vast majority are encapsulated and appe film as smooth rounded or slightly lobulated tumours typically posterior (Fig 172) They are distinguished from intrapulmonary tumours by the induction of an artificial pneumothorax Not infrequently they cause pressure erosion of adjacent ribs and widening of the intercostal spaces Sometimes the intervertebral foramina also are widened and the tumour may extend dumb bell fashion into the neural canal causing pressure symptoms on the cord Such instances are generally ganglioneuromas, but may be malignant A meningioma may produce a similar lesion and several instances of meningocele presenting in the thor

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familial history, neurofibromas elsewhere and skin pigmentation distinguish them In some instances they encircle major vessels or extend into the neck making removal difficult and are much more prone to malignant change than the encapsulated type about 13 per cent undergoing degeneration It has been estimated that a quarter of all neurogenic sarcomas occur in patients having the stigmata of von Recklinghausen's disease

for diagnostic reasons Infected bronchial cysts may give rise to long periods of chronic ill health resembling that caused by severe bronchiectasis, if the lesion is not recognized and removed Furthermore both Moersch and Clagett (and Evarts Graham) have recorded carcinoma developing from a bronchial cyst

An unruptured cyst presents a smoothly round or oval homogeneously opaque shadow on x ray examination At this stage it is difficult to make a positive diagnosis since only by exploratory thoracotomy can it be distinguished from any of the solid mediastinal tumours Bronchoscopy or oesophagoscopy are of little use but aneurysms and oesophageal dilatations are eliminated by radiological means When however the cyst has established a communication its character at least becomes evident since a fluid level will at once be apparent in it The cyst wall is also then visible and is seen to be characteristically thin and regular It is still necessary to differentiate it from a breaking down carcinoma an abscess a diaphragmatic hernia an hydatid or any other cyst And while the fineness and regularity of its wall may make the correct diagnosis probable it is seldom that it can be made certainly This uncertainty, especially when infection has already occurred indicates removal

Histologically bronchial cysts are lined by cuboidal or columnar epithelium, which may or may not be ciliated It is sometimes irregular or even trabeculated and the walls contain characteristically bronchial elements in disorderly arrangement such as cartilage smooth muscle mucus glands and elastic tissue The contents may be clear or viscid and gelatinous fluid, or consist of blood pus and air

Surgical removal is planned through whichever side of the chest seems to afford best access to the cyst and removal presents no particular difficulty except that especial care must be devoted to closing any bronchial fistula

DERMOIDS AND TERATOMAS

No fundamental difference exists between these tumours which share a common origin and a common character In each all three germinal layers may be represented As the name implies however in the dermoid the ectodermal derivatives greatly predominate and the tumour itself is generally cystic The teratoma on the other hand is composed of all constituents and is mainly solid Several theories exist none wholly satisfactory to explain their source but that of Collenberg seems best to fit the facts of their position their nature and their relationship He holds all due to abnormalities in the development of the third and fourth branchial arches at whose union in the foetal midline all the germinal layers are represented in great complexity and from which groups of primitive cells are carried down into the anterior mediastinum by the descent of the heart and great vessels to which they remain intimately related

Be this as it may the teratoid tumours are almost invariably found in the anterior mediastinum only 3 exceptions being known among 250 recorded cases This is so much the case that any tumour of the anterior mediastinum should be regarded as teratoid until otherwise proved Furthermore they are intimately related to the anterior surface of the pericardium and to the great vessels which emerge from it, this constituting one of the chief surgical difficulties in removing them

Simple neurofibromas and ganglioneuromas seldom cause symptoms until they do so by reason of their size, which may become considerable Neurological lesions or pain are generally absent, and many are first detected by routine [illegible]hest radiography Later cough or dyspnoea result as their bulk increases, and [illegible] may not be until the third or fourth decade of life or even later At the same [illegible] cystic degenerative changes are common, with myxomatous and xanthomatous [illegible]nd haemorrhages into the cysts Histologically both neurofibromas and [illegible]euromas are encapsulated, the former being composed of cells from [illegible]th of Schwann, neurones, and much fibrous tissue, the latter of mature [illegible] cells set in a stroma of myelinated and non-myelinated nerve fibres [illegible] change is quite exceptional

[illegible]ically they are best approached by a transpleural postero lateral thoraco-[illegible] unless they are small when a direct extrapleural removal may be [illegible] They are covered on the anterior surface by the parietal pleura, behind [illegible]hey lie and which must be incised Before this is done they appear [illegible]xed to the chest wall, but as soon as the pleura is stripped from them it [illegible]ly easy to mobilize and secure the short pedicle that attaches them to [illegible]r or end of some intercostal space, and either dissect them from the [illegible] sheath or resect, if need be, the nerve from which they grow Bleeding [illegible]ly related intercostal vessels is practically the only surgical difficulty, [illegible] is usually easily controlled Tumours having a dumb-bell extension through in intervertebral foramen into the spinal canal however are not so easily dealt with These generally present with symptoms of cord compression, and a neurosurgical attack as a rule must precede the removal of the intrathoracic portion

BRONCHOGENIC CYSTS

[illegible] arise in any part of the mediastinum but are most often closely related [illegible] to which they may be attached by a pedicle, or to the trachea or a major bronchus If more distally disposed on the bronchial tree they cease to be related to the mediastinum and must be distinguished from other cysts of the lung They originate essentially in a developmental fault, probably by a "pinching off" process from one of the out growing lung buds and may therefore be regarded as first cousins to enterogenous cysts, sequestrated pulmonary segments, or even to dermoids They undoubtedly occur more frequently than published series suggest, although 44 are recorded in the Mayo clinic during the past decade Most have been recognized in the earlier decades of life and very large ones are sometimes seen in infancy, often associated with other developmental abnormalities, and causing respiratory distress and cyanosis More usually they remain symptomless until their presence is complicated by infection, coupled with a fistulous communication with the bronchial tree or with the pleura Persistent cough or dyspnoea may precede this stage, due merely to the pressure effects of the cyst, but once a fistula is established with the bronchus expectoration of the infected contents follows, accompanied by haemoptysis, fever, and of course purulent sputum Rupture of such a cyst into the pleural cavity results in an empyema which will not heal after simple drainage and indeed this accident has been surgically induced by attempts to aspirate cysts mistaken for encysted effusions or

for diagnostic reasons Infected bronchial cysts may give rise to long periods of chronic ill health, resembling that caused by severe bronchiectasis, if the lesion is not recognized and removed Furthermore both Moersch and Clagett (and Evarts Graham) have recorded carcinoma developing from a bronchial cyst

An unruptured cyst presents a smoothly round or oval homogeneously opaque shadow on x ray examination At this stage it is difficult to make a positive diagnosis since only by exploratory thoracotomy can it be distinguished from any of the solid mediastinal tumours Bronchoscopy or oesophagoscopy are of little use, but aneurysms and oesophageal dilatations are eliminated by radiological means When however the cyst has established a communication its character at least becomes evident since a fluid level will at once be apparent in it The cyst wall is also then visible, and is seen to be characteristically thin and regular It is still necessary to differentiate it from a breaking down carcinoma, an abscess, a diaphragmatic hernia, an hydatid or any other cyst And while the fineness and regularity of its wall may make the correct diagnosis probable it is seldom that it can be made certainly This uncertainty, especially when infection has already occurred, indicates removal

Histologically, bronchial cysts are lined by cuboidal or columnar epithelium, which may or may not be ciliated It is sometimes irregular or even trabeculated , and the walls contain characteristically bronchial elements, in disorderly arrange ment, such as cartilage smooth muscle mucus glands and elastic tissue The contents may be clear, or viscid and gelatinous fluid, or consist of blood, pus and air

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DERMOIDS AND TERATOMAS

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Such a position affords little room for expansion since the rigid barrier of the sternum limits it in front, and as these tumours tend to increase in size during adolescence and early adult life, it is then they are apt to cause pressure symptoms, in general throughout the mediastinum, but particularly upon the heart, so that palpitations frequently accompany them They may, however, remain symptomless until overtaken by one or other of the complications to which they are prone Of these, infection is common and occurs readily without any fistulous communication, being presumably blood borne Should it be slight it results only in fever of unexplained origin, and in a formidable increase of those adhesions already binding the tumour to surrounding structures Often, however, it results in rupture into a bronchus or into the pleura with disastrous results Bronchial fistulae on occasion develop without evident infection, so that the contents of a dermoid may be expectorated bit by bit Thus sebaceous material, and particularly hair, may appear in the sputum, confirming in spectacular manner the diagnosis, or it may be aspirated from the pleural cavity or by direct puncture of a cyst Sometimes the tumours present in the neck, or beside the sternum, and either spontaneously or after puncture discharge their characteristic contents upon the surface Pre-sternal dermoids occur, and may be linked dumb-bell fashion with a mediastinal extension, this being sometimes facilitated by sternal defects

The most serious complication of all, however, is malignant change which Rusby (1944) records as affecting 12 9 per cent of all teratoid tumours, the proportion being lower for the more simple dermoid, higher for the solid teratoma, 30 per cent of such malignant growths arise in the former, 70 per cent in the latter Once such a change has occurred there is little or no hope of successful removal

Apart from such pathognomonic symptoms as trichoptysis (the spitting up of hairs), and others resulting from their complications, teratoid growths are diagnosed principally by their radiological appearance This is of a rounded or lobulated shadow, anterior in the mediastinum, bulging to one or other side of the sternum, and in its earlier phases with a clearly defined edge Later on successive attacks of inflammation reduce this sharpness and the edge may become almost as ill defined as that of carcinoma though no invasion has occurred When fistulae are established a fluid level may be seen, and even before this an apparent level called Phemister's line is sometimes distinguished where the fat in such cysts separates at body temperature and floats upon the top of the aqueous content Teeth may be visible in skiagrams of dermoids, or a regular line of punched-out translucent patches representing tooth buds in the midst of a denser germinal ridge Calcification commonly occurs in their walls, but, of course, may also be seen in hydatids, goitres, around tuberculous lesions, or in the pericardium itself (Fig 173) Exploratory puncture should never be resorted to since it may carry infection into the pleural cavity, or even worse, malignant cells The next step in diagnosis must be exploratory thoracotomy

Dermoids usually consist of one cyst, but may be polycystic They contain thick gelatinous mucin and sebaceous matter, and from the inner cyst lining protrude finger-like processes or hard ridges (which may contain cartilage or bone) and from which sprout clumps of lank hair Teeth are often present, as well as other ectodermal derivatives such as neural or retinal tissue They are some-





FIG. 173 (*a* and *b*) A dermoid cyst with marked calcification in its wall. Note the characteristically anterior position in the mediastinum.

(*a*) (*b*)

times attached to the mediastinum by a pedicle, but in any case are adherent to the pericardium, the pleura and the chest wall Accessory dermoids may cluster about the main cyst, having no direct communication with it, or being joined by a narrow stalk

Teratomas share the general character of dermoids but are mainly solid though often having cystic and haemorrhagic areas Their components are more complex representing all layers and often including glandular and skeletal elements

The surgical removal of these tumours may present great difficulty owing to their close and frequently dense and tough adhesion to the anterior surface of the pericardium and the great vessels Such adhesions may be broken comparatively readily, but usually require tedious and dangerous sharp dissection in a region where serious haemorrhage can easily result There is seldom any way of telling before operation whether or not malignant invasion has added to these hazards The aim of surgery, none the less, should invariably be complete excision, and not the marsupialization sometimes practised formerly

TUMOURS OF LYMPHOID TISSUE

Lymphatic tissue is richly disposed in the mediastinum The thoracic duct itself transverses it, lymph from the oesophagus and from the lungs drains into it, and chains of lymphatic glands are found clustered on either side of the trachea, at the carina, about the hila, and in both pulmonary ligaments It is thus not surprising that these mediastinal glands are involved both in localized tumours and granulomas, and in generalized disorders of lymphatic tissue

The commonest cause of local enlargement of the mediastinal glands is without doubt tuberculous adenitis, an invariable, but usually transitory, accompaniment of the primary tuberculous lesion It may however be associated with atelectasis of one or other of the pulmonary lobes from external pressure on a bronchus In later life also, tuberculous adenitis may be mistaken for a primary mediastinal tumour or cyst, and tuberculous glandular masses, sometimes breaking down and semi-cystic, have been removed Such glands are adherent to the neighbouring vessels and bronchi, rendering dissection from the former difficult, and sometimes ulcerating into the lumina of the latter, producing strictures or discharging calcified material into them in the form of "lung stones" or broncholiths The pre-existence of pulmonary tuberculosis, and frequent calcification in the mass, aids diagnosis, but such masses are not necessarily at the hilum, and may simulate cysts or breaking down growths

Metastatic deposits, especially from bronchial carcinoma, are common, and occasionally are more conspicuous than the parent growth On the left side they are often associated with recurrent laryngeal nerve palsy and hoarseness, the only innocent lesion commonly causing this being an aortic aneurysm

Lymphoblastoma and lymphosarcoma occur, the former mainly in children, the latter in young adults, and present smoothly lobulated massive shadows generally responsive to deep x-ray therapy, though lymphoblastomas may not be so, and ultimately fatal When the presence of either is suspected, a diagnostic dose of x-rays provides the best method of identification since they, with lymphadenoma, are practically the only mediastinal tumours which will so respond

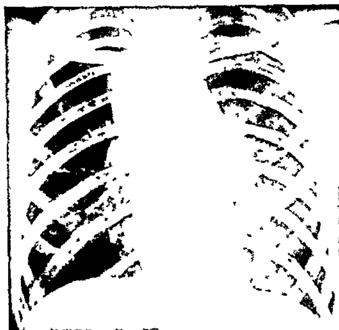


Fig 174 —A case of Hodgkin's disease treated by deep x ray therapy

Reticuloses and Hodgkin's disease similarly treated may be part of a generalized disorder or, not infrequently, appear to be localized to the mediastinum spreading to lymphoid tissue elsewhere only later in the course of the disease and running a comparatively chronic course (Fig 174) Biopsy of accessible lymphatic glands the blood picture, or a Pel Ebstein syndrome may assist diagnosis

Lymphangioma or cystic hygroma rarely occurs in the mediastinum and some reported instances are believed to be confusions with cysts of other origins Arising as a congenital malformation of lymphatic vessels it is uncapsulated multilocular intimately incorporated among the structures surrounding it (so that it cannot be shelled out from them) and lined by flattened mesothelium

PERICARDIAL CYSTS

These not uncommon thin walled cysts, lined by a single layer of endothelial cells, and laxly filled with clear fluid are almost always situated anteriorly and closely related to the cardio-phrenic angle 70 per cent of them are found on the right side (Fig 175) Some have a pedicle attaching them to the pericardium and there are cases on record where fluid in the cyst could be expressed into the pericardium by a narrow communication so that the cyst was in reality a diverticulum On the other hand many have no such attachment and are either loosely related to pericardial fat in the angle or attached to the antero-medial half of the diaphragm That they have a developmental origin is not in doubt but differing theories have been advanced to explain their relationship One suggests that they represent

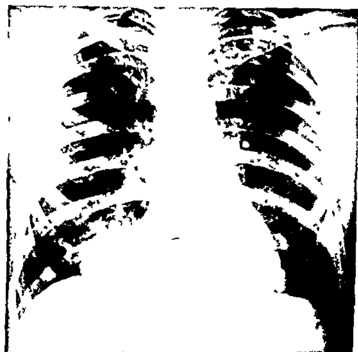


FIG 175—(*a* and *b*) A pericardial coelomic cyst situated typically in the right cardio phrenic angle At operation the cyst was found to be thin walled and contained clear fluid A tenuous pedicle attached it to the pericardium near its junction with the diaphragm

(*a*)



(*b*)

mesenchymal lacunae arising during the formation of the pericardial coelom, another that they are pinched off from the pneumato-enteric recess of the pleuro-peritoneal cavity by the elements of the primitive diaphragm, and constitute the "bursa infracardiaca", a third that they are formed from a constant "ventral parietal recess" of the pericardium during early foetal life.

Be that as it may, the great majority are found in the right cardio-phrenic angle, although they do occur upon the opposite side, and rarely, elsewhere in the mediastinum. As a rule symptomless, they may become sufficiently large to cause dyspnoea, but are usually discovered on routine radiography. Most are flaccid so that their shadow is irregularly convex and not commonly neatly spheroidal. Easily aspirated anteriorly, the fluid contents is as a rule so clear that they have been called "spring-water" cysts. No example of infection or of malignancy has so far been reported. They are easily removed by thoracotomy.

THYMOMA

Some 15 per cent of patients with myasthenia gravis have a thymoma and 75 per cent of patients with a thymoma have myasthenia gravis. No constant relation has been detected between the two, nor has a curare like factor been found in the thymomas, and in a quarter of the patients with a thymoma there is no sign of myasthenia. No constant effect has been observed in myasthenics following removal of the tumour, but Keynes believes that their presence worsens the prognosis, results in greater resistance to Prostigmin and that they should be sought for in every myasthenic patient.

Essentially tumours of the anterior mediastinum, they are usually found in its upper part related to the thymus itself, but may occur much lower down on the pericardium. On lateral x ray films they appear sharply outlined and rounded (Fig. 176), though some show as a flattened bulge on the front of the cardiac shadow. Calcification may be seen in them either in a cyst wall or diffusely scattered.

The tumour itself is slowly growing and composed of thymic constituents, epithelial cells and thymocytes, pallisaded around cystic spaces and blood vessels. Hassall's corpuscles are usually absent. The whole is wrapped in a thick fibrous capsule from which trabeculae traverse the gland and which adheres closely to contiguous structures. In spite of this appearance and whether or not calcification is present, about a quarter of thymomas extend beyond their capsules and become locally invasive rendering removal difficult or impossible. Distant metastases however have not been recorded. In the past they have probably been confused with lymphosarcomas or anaplastic bronchial carcinomas since the small round thymocyte may closely resemble the cells of these tumours.

In general thymomas cause no symptoms unless associated with myasthenia but malignant change may be signalled by pain in the chest, and superior vena caval and innominate obstruction have been described. The onset of substernal pain in a myasthenic is therefore of grave significance and any anterior mediastinal tumour in a myasthenic is virtually certain to be a thymoma.

Surgical access is best gained by splitting the sternum down to the third or fourth interspace. The thymus lies directly beneath between the right and left

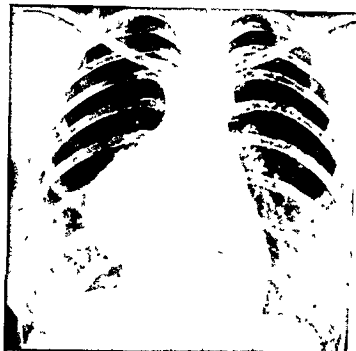


FIG 176 —A malignant thymoma in a girl of 19 years without myasthenia It was however still removable although this involved resection of part of the middle lobe all the anterior pericardium some of the opposite pleura and the thymus gland

pleurae, and can be removed largely by blunt dissection, and ligation of its main venous communication with the left innominate vein Should surgical removal of an invasive thymoma prove impossible, deep x-radiation is worth trial since some tumours are responsive

ENTEROGENOUS CYSTS

Most of the reported cases have been in infants under 1 year of age, suggesting that their presence is not long compatible with life , but a few examples are found in older groups They have a similar origin to bronchial cysts, arising as diverticulae from the developing foregut during foetal life, and are characterized by the possession of a mucosal lining resembling some portion of the gut, but especially and principally the stomach This may be complete, and secrete gastric juice copiously with all its components, so that the cyst resembles an accessory but isolated stomach , or represented by such small fragments of gastric mucosa that its identity may pass unrecognized and the cyst contain little but mucus As is the case with most congenital anomalies, other malformations may co-exist, such as cysts and diverticulae elsewhere, or spina bifida

Generally the cyst is closely related to the oesophagus and so is found posteriorly, and in nine cases out of ten upon the right side — possibly because of the presence *of the aorta on the left It may be densely adherent to its parietes, and the wall is* sometimes as thick as that of the stomach and includes submucosal and muscular

layers. Such cysts are found only in infants since the gastric juices they secrete cause them rapidly to distend and either rupture into the oesophagus, lung, or pleura, or cause death by interfering with respiratory function. Those seen in later life, while also occupying a posterior position will contain little secreting mucosa and have a thinner wall. Seven examples of gastric ulceration within enterogenous cysts are recorded, and 4 have penetrated into bronchi.

In infancy cough, dyspnoea, and cyanosis occur, due to the cyst's bulk or collapse of the adjacent lung, and haemoptysis, haematemesis, and melaena are all described depending upon the course taken by the ulceration. Once fistula is established a fluid level will be seen across the x ray shadow, revealing its cystic character. Should aspiration have been carried out under the impression that an effusion was being dealt with the presence of hydrochloric acid or pepsin in the fluid withdrawn make the diagnosis clear, though this measure is most unwise. Cysts containing little or no gastric mucosa remain symptomless. Surgical removal is life saving in infancy, and advisable at any age.

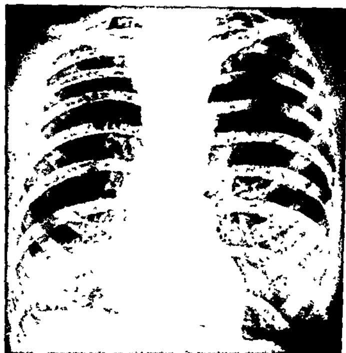
INTRATHORACIC GOITRE

Colloid goitres frequently become substernal occupying a position in the anterior part of the superior mediastinum and may here be mistaken for any of the tumours or cysts intrinsic to the region in particular those arising from the thymus. They may attain great size but retain from the nature of their growth their vascular attachments in the neck. In time most undergo toxic degeneration with resultant tachycardia or auricular fibrillation. From their situation in the rigid and constricted thoracic outlet pressure signs are often in evidence with dilatation of neck veins and compression of the trachea which is displaced to one or other side according to the thyroid lobe principally enlarged or flattened from in front. Symptoms as well as evidence of toxicity may include an irritative cough, dyspnoea or dysphagia. Should haemorrhage occur into one of the cystic spaces of the gland acute exacerbation of symptoms is apt to result.

Clinically such patients may have evidence of thyroid enlargement in the neck, or the scar of previous thyroidectomy, but sometimes the enlargement is wholly substernal. It is usually possible however to palpate the upper lobe of the tumour in the suprasternal notch or supraclavicular fossa, especially on deep inspiration, and it characteristically rises on deglutition distinguishing itself thus from other intrathoracic growths.

In antero posterior skiagrams of the chest an irregularly rounded shadow is seen in the superior mediastinum, generally more prominent upon one side and displacing the trachea to the other. Its convexity is downwards, and its upper limits cannot be defined, shading off into the base of the neck. Lateral views show it to be anteriorly and often reveal calcification which may be extensive (Fig. 177). The use of radioactive iodine isotopes is now increasingly used to identify intrathoracic thyroid masses.

The surgical approach should always be by means of an orthodox thyroidectomy collar incision in the neck, since it is here the vascular pedicle is to be secured. If necessary the infra hyoid muscles on one side are divided and dissection carried down to the capsule of the gland. The thyroid vessels are secured and the mass



(*a*)

FIG 177.—Retrosternal thyroid (*a*) showing the tumour chiefly to the right of the mediastinum, (*b*) lateral film shows its anterior position and reveals considerable calcification in its substance



(*b*)

delivered into the neck by a combination of gentle traction and leverage from below, on occasion with a spoon Some surgeons recommend evacuating the colloid material from the interior of larger glands but all are agreed that sternum splitting is seldom if ever required Subtotal thyroidectomy is then performed care being taken not to damage the recurrent laryngeal nerve as it courses on the postero-lateral surface of the glands True ectopic intrathoracic thyroids (having no vascular connexions with neck) have been recorded but are exceedingly rare They are diagnosed and can be removed, only by exploratory thoracotomy

RARER TUMOURS

Such then are the commoner tumours encountered in the mediastinum a variety of rarer growths remain Among them are found lipomas 40 of which have been recorded All are extrapleural and may attain great size being signalled by a progressive and ultimately severe dyspnoea Most remain wholly within the thorax but 9 have presented dumb-bell fashion beside the sternum and 7 in the root of the neck, where they are felt to be soft lobulated and to transmit an impulse on coughing X ray appearances are those of an opacity of which the edges may be perceptibly more translucent than the centre but which otherwise resembles an encysted or interlobar effusion

Some dozen examples of meningocele protruding into the chest are described most of them at first mistaken for neurofibromas because of their smooth outline and posterior situation They may cause pressure symptoms and bone erosion of intervertebral foramina and might be diagnosed (were their nature suspected) by myelography The writer has also seen a meningioma presenting in the upper part of the chest

Haemangioma of the mediastinum may be single and encapsulated in multiple foci as part of generalized congenital telangiectasia or frankly malignant At least 17 instances occur in the literature of which 8 were malignant and 2 were part of many primary lesions Appearing anywhere in the mediastinum they are not to be identified before operation though a vascular bruit may be audible over them This feature however they share with any very vascular tumour with aneurysms, and with arteriovenous communications

Other, and more exceptional tumours numbered only in ones or twos but appearing from time to time still further to complicate the already difficult differential diagnosis of mediastinal neoplasms are leiomyoma an innocent growth associated with the smooth muscle of the oesophagus parathyroid adenoma with the skeletal changes raised serum calcium and generalized osteitis fibrosa plasmocytomas single or multiple innocent or malignant fibroma chondroma myxoma , and xanthoma

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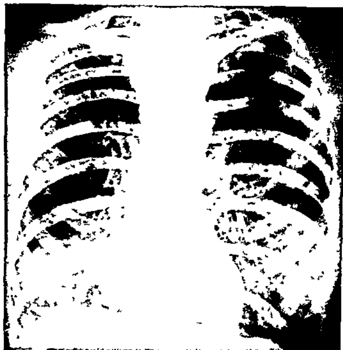


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(*a*)



(*b*)

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CHAPTER 30

DISEASES OF THE DIAPHRAGM

KENNETH M A PERRY

UPWARD displacement of the diaphragm may result from the pressure from below of ascites, pregnancy and abdominal tumour or from pull from above by pulmonary fibrosis or collapse In all these cases, however, it moves normally When paralysis is present, there will always be paradoxical movement visible on screening

DIAPHRAGMATIC PARALYSIS

The phrenic nerve is sometimes divided as a therapeutic measure If it is crushed paralysis will last for 6 months, if it is avulsed the paralysis will be permanent Trophic changes in the diaphragm follow as early as 3 days after paralysis and atrophy is well established after a month The muscle is reduced in thickness, fat is deposited and fibrous tissue develops The phrenic nerve may be divided by trauma or disease processes involving the peripheral course of the nerve, spinal cord or medulla Thus diaphragmatic paresis may occur in acute anterior poliomyelitis, tumour, progressive muscular atrophy, myelitis, haemorrhage and injuries and caries of the cervical vertebrae Peripheral lesions of the phrenic nerve include neck wounds, destruction by neoplastic masses, diphtheritic and other types of peripheral neuritis and avulsion from birth injury as an extension of Erb's paralysis Blattner (1942) and Light (1944) have recorded cases in newborn infants resulting from intra-abdominal malposition Mediastinal tumours, and chronic inflammatory processes in the mediastinal and paratracheal lymph nodes not infrequently involve the phrenic nerve (Fig 178) Friedman (1950) recorded 6 cases of phrenic paralysis in association with pneumonia Phrenic paralysis is occasionally found in patients when no explanation can be given for its development but it is extremely doubtful whether stretching of the nerve by aortic aneurysm ever causes paralysis though Sanguinette and Galzerano (1943) have suggested that this may happen

EVENTRATION OF THE DIAPHRAGM

Eventration of diaphragm is the name applied to a condition in which the diaphragm is abnormally high, as contrasted with an upward displacement from pathological causes (Fig 179) It is a rare condition and is commoner on the left side than the right (Korns, 1921, Reed and Borden, 1935, Evans and Simpson, 1950) The condition is probably of congenital origin but may result from birth injury to the phrenic nerve The diaphragmatic muscle is usually atrophied and replaced by a related translucent membrane of white fibrous tissue with few muscle fibres It is frequently paralysed and shows paradoxical movement, but this is not always so The condition does not as a rule cause symptoms and in the rare cases where they do occur they are usually of a dyspeptic nature Shortness of breath on

exertion and cough and pain in the left side may occur *Physical examination* will reveal poor movement of the left side of the chest and its paradoxical nature may be evident Fluoroscopy will confirm these findings while the use of a barium meal will differentiate the condition from a short oesophagus with herniation The stomach often appears as a characteristic inverted fish hook, being rotated so that the greater curvature is uppermost under the dome of the diaphragm, the pylorus and pyloric antrum lie anteriorly in the left upper quadrant of the abdomen (Rosenfeld, 1944) A pneumoperitoneum will assist in outlining the diaphragm

Treatment is not satisfactory Dyspepsia can be treated with a light diet phenobarbitone, and alkaline and antispasmodic mixtures Surgery has been used in the treatment of the condition the diaphragm being plicated or incised, overlapped and sutured At the operation the appearance and calibre of the phrenic nerve is always normal

DIAPHRAGMATIC TRICHINIASIS

Trichiniasis is caused by eating pork or bear meat which contains living larvae of the nematode *Trichinella spiralis* These larvae are liberated in the gastro intestinal tract during the digestion of the meat They quickly mature and then mate, when the female produces new larvae which penetrate the wall of the gut to enter the blood stream Some are deposited in the muscles where they remain alive for years and become encysted others lodge in other organs where they die The human illness occurs only during the first few weeks of infestation

Symptoms rarely arise during the stage of intestinal invasion and there are no symptoms after the larvae become encysted These usually arise 7–12 days after ingestion and consist of fever, facial oedema sometimes oedema elsewhere skin rashes and muscular pains Chest pain is common and is attributed to infestation of the intercostal muscles and the diaphragm There is sometimes hiccough cough is also common and is unproductive, probably resulting from the presence of parasites in the lungs haemoptysis occurs occasionally

Eosinophilia is the best guide to early diagnosis The parasites can sometimes [illegible]

trichinella antigen are useful diagnostic procedures

The condition quickly resolves and little is required in the way of treatment The disease can readily be prevented by ensuring that pork is well cooked An outbreak occurred in Wolverhampton in 1941 as a result of people eating uncooked sausage meat (Sheldon 1941)

DIAPHRAGMATIC HERNIA

There are two types of diaphragmatic hernia—those due to trauma and those resulting from congenital weakness Traumatic herniae may either be due to penetrating or non penetrating injuries or be a post operative occurrence (Fig 180) Where they result from non penetrating injuries, the injury is usually of a crushing nature but it may be by less violent trauma such as falling downstairs In the case of penetrating or post operative injuries the cause is always

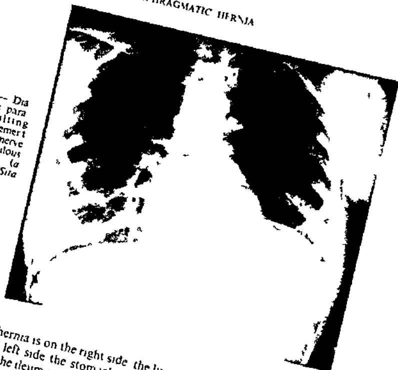
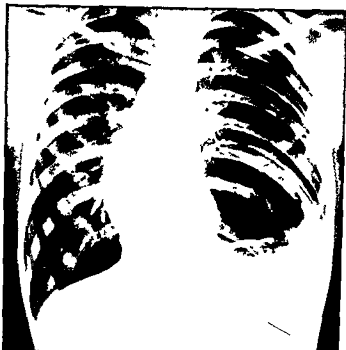


Fig 178 — Diaphragmatic paralysis resulting from involvement of phrenic nerve in a tuberculous adenitis (a patient of Dr Sita Lumpden)

evident When the hernia is on the right side the liver and colon are the herniated viscera while on the left side the stomach and colon are herniated and perhaps if the rupture is large the ileum and spleen as well (Figs 181–182) Non traumatic diaphragmatic herniae may either occur through the oesophageal hiatus or through defects in the muscular and tendinous portions of the diaphragm The latter are more common on the left side than on the right They can occur through the foramen of Bochdalek the foramen of Morgagni through a congenital absence of the posterior fourth or through defects in various parts of the dome On the left side they are most commonly posterior on the right side where the herniae are often parasternal they are usually anterior and contain colon or liver The oesophageal hiatus herniae may be associated with a short oesophagus oesophagitis and peptic ulceration or with a normal oesophagus when they may be of the para oesophageal or sliding gastric oesophageal type They develop in adult life as a result of obesity and laxity of the tissues at the hiatus from advancing age or increased intra abdominal pressure Oesophagoscopy will show if oesophagitis or peptic ulceration is present

In infancy shortness of breath cyanosis colicky pain vomiting and abdominal distension are the principal symptoms These are often improved by holding the child in the upright position thus enabling the hernia to reduce itself In later life the symptoms are usually dyspepsia or retro-sternal pain resembling that caused by coronary ischaemia Hiccough is an occasional symptom

Screening the patient in the recumbent position after a barium meal is the most important aid to diagnosis In right sided cases a barium enema is most helpful

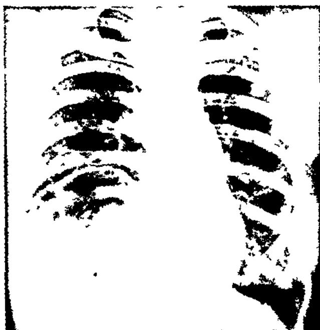


(a)

FIG 179 — Typical example of eventration of the left side of the diaphragm (a) Postero anterior view, (b) lateral view An example of the rare right sided eventration is seen in (c) and (d) (c) Postero-anterior and (d) lateral



(b)



(c)



(d)

Skiagrams usually show the abnormality, which has to be differentiated from cysts and tumours of surrounding structures or from pleural effusion

If a hernia is liable to strangulate, surgical repair is indicated Treatment of hiatal herniae should be medical and the peptic ulcer régime used if oesophagitis is present The patient should sleep propped up Left phrenic paralysis gives good results in older patients but repair of the defect by the transthoracic approach is usually the best treatment If stricture or severe ulceration is present, dilatation of the oesophagus with application of silver nitrate or resection of the affected portion may be necessary

SUB-DIAPHRAGMATIC ABSCESS

This is not truly a disease of the chest but it so often causes sympathetic lesions above the diaphragm that a book of *diseases of the chest is not complete without* some consideration of the condition There are five spaces in which these abscesses may form, right and left supra-hepatic are the commonest sites, the others being right infra-hepatic and left anterior and posterior infra-hepatic, the frequency being in that order The supra-hepatic lies between the diaphragm and the dome of the liver while the infra-hepatic space is the under surface of the liver and the transverse colon The supra-hepatic space is divided into right and left portions by the falciform ligament and the infra-hepatic is similarly divided by the ligamentum teres and ligamentum venosum

Most cases of subdiaphragmatic abscess arise from pocketing of pus following a *general peritonitis resulting from appendicitis, perforation of peptic ulcers, or* malignant neoplasms of stomach or colon, cholecystitis and liver abscess In the last mentioned case there is a high mortality rate The majority of these occur post-operatively Some may result from direct spread of the pyogenic infection, but *there* is little evidence to confirm that they can result from lymphatic spread as was once thought Occasionally they occur as part of a pyaemic process and rarely from a spread from inside the chest They may occur at any age but are [illegible]

effusions are usually sterile, but empyema [illegible] *quarter* of the cases (Harley, 1949) and suppurative pneumonitis also results in a few Perforation of the diaphragm either from spread of the infection or as a result of transpleural aspiration or damage is the most common cause of the suppurating conditions

Clinical features

The clinical manifestations of this disease tend to appear 3–4 weeks after the causal laparotomy Faxon (1940) in reviewing 111 patients from the Massachusetts General Hospital found an average interval of 26 days The usual presenting symptoms are fever and pain along one of the costal margins The onset is sometimes abrupt with rigors, sweating, vomiting, abdominal pain and collapse More often it is subacute and in a few it is slow and insidious and *in this last group it*

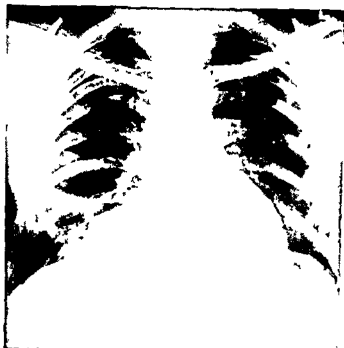


FIG 180 — Trau mat c hern a of l er and colon through r ght s de of d aphragm (b) L p odol n bronchus and bar ium n colon (a pa ı en (f D S aLı p l n)

(a)



(b



(a)

FIG 181 —D aphragm atic hern a of colon through an anter or parasternal defect in the r ght side of d a phragm (b) lateral v ew showing bar um in colon



(b)



FIG 182 —D aphragm atic hernia of stomach and intes t nes through the left d aphragm

may go unrecognized for many months Other symptoms which may occur include cough shortness of breath pain in the lower part of the chest or generalized toxaemia

The physical signs are scanty unless a pleural effusion is present when there will be dullness and absent breath sounds over the lower part of the chest Other wise localized tenderness over the site of the abscess will be the only sign that can be elicited A blood count will always reveal a polymorphonuclear leucocytosis Diagnostic aspiration is inadvisable owing to its unreliability and because of the danger of infecting the pleura The most significant radiological finding is fixation and elevation of the diaphragm and on the right side if the diaphragm is normal in position and moves freely on resp ration a diagnos s of subdiaphragmatic abscess can be excluded This rule however is not absolute on the left side When air is present under the diaphragm it is a valuable aid to diagnosis but th s occurs in only about a quarter of the cases on the left s de it must be distinguished from gas in the stomach In about 25 per cent of cases a pleural effusion is present and this will be evident in the skiagrams as will pulmonary consolidation if it is present It is important to take lateral pictures as well as antero posterior ones while postero-anterior ones in lateral recumbency are sometimes helpful

Treatment

Chemotherapy with adequate dosage of sulphonamides penicillin and streptomycin will prevent the development of a large number of sub-d aphragmatic

abscesses, and make the condition a rare one Chloromycetin and terramycin will also play a part in achieving this

Once there is an established abscess surgical drainage becomes essential and the mortality rate is greatly reduced if an extra serous method of drainage is adopted An incision can be made over the twelfth rib which is resected sub periosteally The abscess can then be drained by a transverse incision through the bed of the rib at the level of the spinal process of the first lumbar vertebra Ochsner and DeBakey (1938) give a mortality rate of 20 8 per cent for retroperitoneal drainage compared with 36 2 per cent for transpleural and 35 1 per cent for transperitoneal and Harley (1949) gives 11 per cent for extra serous drainage and 33 per cent for trans serous

Serous pleural effusion will usually absorb spontaneously when the abscess has been drained but intrathoracic suppuration usually makes the condition more serious When the abscess is drained, however, by the establishment of a bronchial fistula *the mortality is significantly diminished*

DIAPHRAGMATIC TUMOURS

Primary cysts and tumours are rare They were first described by Grancher (1868),

the condition was carefully reviewed by Cruickshank and Cruickshank (1951) These congenital cysts appear to be of mesothelial origin (Fig 183) They arise from that part of the coelom which becomes the pleuro pericardial canal and are closely related to pleuro pericardial cysts Many are situated within the substance of the diaphragmatic muscle Histologically they have a fibrous wall and are *lined by a single layer of flattened cells*

Tumours of various kinds occur in the diaphragm , sarcomas, which have been described as rhabdomyosarcoma, fibromyosarcoma, mixed cell sarcoma myo blastic sarcoma leiomyosarcoma, myosarcoma and endothelial sarcoma, are the *commonest Lipomas are the next most frequent tumours , but fibromas,* mesotheliomas, neurofibromas fibromyomas, chondromas angiofibromas and haemangio endotheliomas have been described Endometriomas may also occur in the diaphragm

Severe pain in the lower part of the chest aggravated by deep breathing is the commonest symptom Cough with a little mucoid sputum occasionally streaked with blood has been reported Arkless (1942) described the coincidence of a rhabdomyosarcoma of the diaphragm idiopathic hypoglycaemia and retroperitoneal sarcoma *On examination a tumour is sometimes palpable between the lower* ribs or in the upper abdomen It moves with respiration It has on occasion been mistaken for the liver or spleen Skiagrams will show a tumour in the lower part of the chest which is difficult to separate from surrounding structures It *moves synchronously with the diaphragm Diagnostic pneumothorax on pneumo* peritoneum may help to define its situation Thoracoscopy may also help in the diagnosis

Prognosis depends on the nature of the tumour, in the malignant conditions it *is always bad*



FIG 183 — Mesothelial cyst of the diaphragm lying in the left costo diaphragmatic angle (b) lateral view of same (*a patient of Mr G Flavell*)

(*a*)



The cysts and tumours should always be removed surgically, since more than half are malignant and it is only after removal that the diagnosis can be finally established

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CHAPTER 31

ANAESTHESIA IN CHEST DISEASE

A I Parry Brown

The patient with chest disease who needs to be anaesthetized presents two problems to the anaesthetist The anaesthetic may aggravate the chest disease, or the diseased and inefficient respiratory apparatus may make the administration of the anaesthetic more difficult and dangerous *The first important step is to establish* the extent of the respiratory disease The site of the lesion, the likelihood of sputum, and the response to treatment can be ascertained on reference to the medical history The ease with which the respiratory exchange is maintained and the reserve for exercise are points of value in planning the method of anaesthesia

SURGICAL EMERGENCIES

First there is the surgical emergency with acute respiratory disease There is no

to effect a transfer The vital capacity may be reduced by congestion or effusion, or even by previous treatment, as by the use of an artificial pneumothorax Additional barriers may be interposed between the ventilating gases and the circulating blood as, for example, widespread mucus in a tracheo-bronchitis Air may be drawn into areas of lung without a corresponding blood supply as in the case of a giant air cyst or in chronic emphysema These patients become sub-oxygenated on mixtures which would be quite adequate for the normal patient The anaesthetic is less quickly absorbed and excreted so that both the induction and the recovery period are prolonged

The patient who knows that he has a chest disease fears that the anaesthetic will affect him adversely This is particularly true in patients with pulmonary tuberculosis Such patients when faced with an operation for acute appendicitis are genuinely, perhaps with reason, more frightened of the anaesthetic than of the operation This state of nervous tension makes the induction more difficult and must be allayed by reassurance, which can only come from a careful pre-operative assessment

In this group of surgical emergencies time is not available for any thorough

limited in serious chest disease If the patient is dyspnoeic or orthopnoeic position on the table will be intolerable Many of the techniques of local analgesia *used for abdominal operations* involve the paralysis of the intercostal muscles,

which would increase the respiratory embarrassment. Cases with uncontrollable coughing are difficult to manage at the time of operation if spinal analgesia is chosen. They are subjected to a greater than normal risk of post-spinal headache, for the persistent coughing encourages the leak of cerebrospinal fluid through the dural puncture. If some of these points contra-indicate the use of local analgesia then cyclopropane is probably the best of the general anaesthetic agents being non-irritant to the respiratory tract and the administration is with high oxygen concentrations. The respiration is quiet under its action; a rapid return of the protective reflexes after the operation follows as cyclopropane is quickly eliminated.

A woman was admitted to hospital suffering from lobar pneumonia, who had been in labour for 24 hours and had reached the stage of exhaustion. She was cyanosed and orthopnoeic with a respiratory rate of 40 per minute. It was obvious that she was unable to deliver herself and as the cervix was nearly fully dilated it was decided to deliver her with high forceps. After premedication with atropine, anaesthesia was induced with a mixture of cyclopropane 20 per cent and oxygen 80 per cent. On this mixture her respiratory distress was relieved, and her colour improved. As she lost consciousness it was possible to lower her into the horizontal position without a return of the cyanosis. High forceps were applied in the lithotomy position and the woman was safely delivered. The return of consciousness was rapid and as the splinting of the diaphragm by the uterus was relieved she was able to breathe easily and made a rapid recovery under medical treatment.

Whilst such a case suited the technique almost as well as the technique suited the case it must be remembered that these cases can be difficult and it is unwise to use an unfamiliar technique. It is safer to use a routine method, provided that it is physiologically sound, than to use an agent which however ideal suffers the disadvantage of unfamiliarity. Nitrous oxide, oxygen and ether provides a sequence which although humdrum is safe during the administration and which carries little extra risk of post-operative complications if steps are taken to prevent the spread of secretions whilst the reflexes are inactive and ciliary movement is reduced.

An even more dangerous error than using an appropriate anaesthetic agent but with insufficient experience is to strain a weak agent beyond its proper capabilities. Nitrous oxide has great advantages as an anaesthetic agent for it is non-toxic, and rapidly eliminated, but it is weak and can only produce more than the light planes of anaesthesia by depriving the patient of oxygen. The old finding of "fit for gas" has no place in anaesthesia for cases with chest disease.

SPREAD OF SECRETION

Unsound technique greatly increases the danger of the spread of secretions. Forced *hyperpnoea by the use of* carbon dioxide may simplify and accelerate the induction of anaesthesia in the healthy patient, but it is obviously inapplicable in these cases. The cilia of the respiratory mucosa are easily damaged. Rough instrumentation may destroy them and leave the patient partially deprived of this mechanism for the removal of secretions. The cuffed endotracheal tube particularly when lubricated by a greasy ointment containing a powerful surface anaesthetic is injurious and should only be used when essential. The spraying of the larynx

and bronchial tree with local analgesia solutions carries with it the danger that secretions may spread whilst reflexes are depressed, and if it is necessary to use them steps must be taken to control secretions In all except acute emergencies this control can start pre-operatively Postural drainage and suitable medical treatment can reduce the amount of secretion so that the risk of flooding new areas of lung is greatly reduced Premedication with atropine or scopolamine by keeping the respiratory tract clear from mucus reduces the volume of any secretions and so the likelihood of soiling, but at the price of making viscid plugs which are harder to cough up

Control of spread

The secretions may be controlled during the operation by suction by posture, or by the isolation of the diseased area of lung The removal of secretions by suction is the most immediately attractive solution, for if the anaesthetic gases are being given through an endotracheal tube the only change from normal routine is the occasional passage of a suction catheter down the endotracheal tube The disadvantages are that it interferes with the smooth administration for, the anaes thetic gases are lost during the periods of aspiration It requires deeper anaesthesia to prevent coughing when the end of the suction catheter touches the carina than is needed for many operations

It may fail because of the silent spread of small quantities of sputum or because large volumes need such constant attention that no time is left to give the anaesthetic This blind suction may make worse the condition in the lung If the catheter is passed into small bronchi all the air may be drawn from a segment which will fail to re-expand Haemorrhage may be caused particularly if the disease is an adenoma or carcinoma of the bronchus Despite these disadvantages intrabronchial suction remains a most valuable method of controlling secretions and of preventing the contamination of fresh areas of lung It should be applied at intervals determined by the amount of secretion and at stages known to be associated with the release of sputum as for example when the patient is moved on to the operating table The relative lengths of the endotracheal tube and of the suction catheter should be known so that the position of the sucker may be estimated

A suitable position on the table may eliminate the danger of inhalation of sputum The secretions may be retained in the diseased area where they cause no reflex disturbance or interference with respiration, or a position can be chosen in which any sputum that is released will run up towards the mouth rather than be absorbed in new areas of lung

The former is useful in short procedures

with the child half sitting up and partly on his left side In this position the left lower lobe is dependent and secretions were retained in the diseased area Nitrous oxide oxygen and ether were used and a smooth induction was obtained without coughing When the child was fully anaesthetized a naso tracheal tube was introduced and through

this a fine gum-elastic suction catheter was passed Nearly an ounce of sputum was aspirated, although it had caused no disturbance whilst retained in the diseased area of lung The suction catheter was removed and the operation proceeded normally

Protection is possible by the postural method if the operation is performed in the prone position or in the lateral position if a Trendelenburg tilt is used, but in the supine position, unless an extreme head down tilt is used, the secretions will not gravitate towards the mouth

The control of secretions by endobronchial tamponade is not usually applicable to cases undergoing general surgery but rather to cases for lung surgery where the diseased area will be removed, for the mucosa at the site of the obstruction will be injured and further damage may be done in the isolated area of the lung

This danger from the inhalation of secretions is not limited to the duration of the operation but continues throughout the subsequent period of depressed reflexes The patient may not have such constant supervision during the recovery period so that a technique which allows a quick return to full reflex activity will be advantageous The worst anaesthetics in this respect are ether, when the anaesthesia is deep, and the short-acting intravenous barbiturates, when repeated administration is needed to maintain anaesthesia for long periods The advisability of increasing the rate of excretion of ether by the inhalation of carbon dioxide and oxygen at the end of the operation should be considered but there is a danger that this forced over breathing may spread secretions into small bronchi which they will occlude This method can give a false sense of security after a long deep ether anaesthesia, for during the period of over-ventilation the concentration of ether in the blood is lowered and reflex activity returns There is afterwards a reaction when the breathing is very shallow, during this period ether is mobilized from body tissues and the blood concentration rises again

DANGER OF COLLAPSE OF LUNGS

Diminished movement of the chest wall may lead to collapse of the underlying lung This restricted movement may be due to pain as after any upper abdominal operation, or due to paralysis of some of the respiratory musculature Reference has already been made to this undesirable side-action of spinal analgesia The muscle relaxants which spare the diaphragm but paralyse the intercostal musculature produce paradoxical movement of the apices of the lung, which is inefficient and likely to cause sub-oxygenation in patients with respiratory disease The artificial respiration which will correct this is likely to spread secretions unless adequate steps are taken to control them

AGGRAVATION OF EXISTING DISEASE

Besides these local effects an anaesthetic can aggravate pre-existing diseases by its general toxic action In pulmonary tuberculosis a critical balance may be maintained between the invasive powers of the organism and the resistance of the host If this resistance is lowered, as it may well be by an operation, an exacerbation of the disease is to be expected The lowered resistance is in part due to surgical trauma, in part to blood loss and in part to the anaesthetic The anaes

thetist can help to reduce the trauma by providing complete muscular relaxation, and some general effects of the trauma can be reduced by blocking painful stimuli with local analgesia The careful replacement of blood loss is especially worth while in these cases An estimate of the blood loss as by weighing the used swabs helps in assessing the volume of the transfusion required for the risk of causing pulmonary oedema by over transfusion is greater with pre-existing respiratory disease Those drugs which interfere with the metabolic processes of the body are most likely to lower the resistance of the patient On this count the prolonged deep administration of ether or chloroform should be avoided, but it is only fair to add that some authorities use ether with excellent results for all thoracic operations including those for tuberculosis Prolonged sub oxygenation, or bouts of anoxia from " incidents " during the anaesthetic are probably worse

CHOICE OF TECHNIQUE

The chest disease may make some ordinary anaesthetic techniques unsuitable The

respiration cease to work as consciousness is lost and respiratory arrest may follow In such a case a preliminary use of cocaine in the respiratory tract will facilitate immediate intubation, and as the endotracheal tube passes the obstruction it gives immediate relief It is sometimes difficult to pass the obstruction without entering the right bronchus If this occurs it becomes much easier for air to enter the right than the left lung This causes differences in pleural pressure on the two sides and mediastinal shift results

The fear of spasm is ever present in patients who give a history of asthma, and an induction with thiopentone may precipitate a severe laryngospasm Fortunately ether is a reliable agent for such cases Instead of more coughing and spasm as might be expected, the ether relaxes the bronchial musculature and the breathing ... Indeed there is a marked relief of ... -indicated by ... the adminis-tration of cocaine to the respiratory tract before the induction of anaesthesia, or by the use of an intravenous transfusion with a procaine solution

PROBLEMS PRESENTED BY PNEUMOTHORAX CASES

The patient with a pneumothorax may present difficulties If the proposed operation requires the patient to lie in the lateral position with the pneumothorax undermost, the rising diaphragm on the underside and the sagging mediastinum may cause the pleural pressures in the artificial pneumothorax to rise so high that ... In non- ... Too often ... rax has a larger than usual refill pre operatively in the hope of minimizing the effect of the anaesthetic on the underlying lung

ADDED PROBLEMS IN CHEST OPERATIONS

If a chest operation is proposed some of the problems particularly those of the control of the secretions and the maintenance of adequate ventilation become more urgent and special techniques must be used Most chest operations involve an open pleura and in all paradoxical respiratory movement is likely to occur in areas of lung unsupported by a rigid chest wall

This paradoxical movement makes respiration inefficient for some of the effort of inspiration draws air from the paradoxically moving portion of the lung whilst in expiration a proportion of the air which should be expelled from the trachea will distend the unsupported lung This pendulum air takes no part in the respiratory exchange and may be the vehicle for dispersing secretions through previously healthy lung

The mechanism of this paradoxical movement is that unsupported lung moves in response to pressure changes in the bronchi This pressure is high during expiration and less than atmospheric during inspiration These pressure differences are small with quiet unforced breathing A patient well premedicated and free from anxiety who receives a careful local analgesia of the chest wall will breath so quietly that the area of pleura exposed in a thoracoplasty for tuberculosis will move very little If the patient becomes tense perhaps from inadequacy of the local analgesia the breathing becomes strained and paradoxical movement becomes apparent The effort of working the valves of an anaesthetic machine increases the pressure differences so it is difficult to give a light general anaesthetic to cover any deficiencies in the local analgesia without causing marked paradoxical movement

Nevertheless local analgesia must be accepted as a most useful technique for operations on the chest wall most notably for thoracoplasty and for the drainage of empyemas

This rhythm of pressure changes can be reversed if with the patient in an apnoeic state respiratory exchange is maintained by an intermittent positive pressure anaesthesia This controlled respiration ensures that all areas of lung fill during inspiration and empty in expiration thus eliminating the pendulum air with its effect of a greatly increased respiratory dead space

The use of controlled respiration is not essential for chest surgery but it greatly increases the scope of surgery by keeping the patient fit and well oxygenated and by providing quiet conditions in which to operate It does however demand a more careful control of secretions for the positive pressure of inspiration could easily drive secretions into small bronchioles which would be obstructed In overcoming this difficulty besides the methods already outlined of posture and intra bronchial suction resort is often made to bronchial tamponade

The anaesthetic may be given into one main bronchus the other lung being isolated from the anaesthetic circuit by an inflated balloon which encircles the tube This is carried out more easily if the tube can be placed on the left side for the left main bronchus can accommodate the balloon and allow free drainage from the right lung outside the anaesthetic tube When the endobronchial tube is passed on the right side the balloon must be partly in the trachea for the distance from the carina to the orifice of the right upper bronchus is too short to take the balloon

As a result secretions are retained in the left lung, which threaten catastrophe should at any time the tube become displaced The movement of these endo bronchial tubes must always be feared The position of the inflated cuff must be accurate to within a centimetre The tube is fixed at the proximal end During dissection traction is made on the hilum of the lung, and the tube becomes dis placed into the trachea

An alternative is to obstruct the bronchus of the diseased lobe or lung with a balloon which encircles a fine drainage tube, whilst the anaesthetic is given through an endotracheal tube into the remainder of the lung These drainage blockers give good control of secretions and allow the diseased area of lung to collapse during the operation There are two commonly used types of blocker The

net gives a better adhesion than rubber to the mucosa of the bronchus This balloon is bulky and can only be passed through an adult 11 millimetre broncho-scope The Magill blocker is more delicately made and can be passed through a smaller bronchoscope which gives it a wider applicability, but more skill is required to prevent its becoming displaced during the operation These drainage blockers can be used for pneumonectomies, and lower lobectomies on either side, but are not reliable for upper lobectomies as it is only occasionally possible to introduce them into the upper lobe orifice

under lung The removal of secretions is completed by intermittent intra bronchial suction, and by a post-operative bronchoscopy

If the secretions are controlled, and paradoxical movement is prevented by the use of controlled ventilation, the anaesthesia required for thoracic surgery is simple An induction with thiopentone and maintenance with nitrous oxide and oxygen will suffice if curare is given to allow the over ventilation necessary to

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the patient appears to be of value in preventing post operative respiratory complications

SUMMARY

To summarize, it may be said that local analgesia should be considered if it is suitable for the operation proposed and if respiratory distress is not a marked feature of the disease Routine general anaesthesia is safer than experimenting with new methods and in particular ether despite its irritant properties is safer than the injudicious use of the short acting barbiturates In acute respiratory infections with dyspnoea cyclopropane is valuable to those experienced in using it Light general anaesthesia used with muscle relaxants is almost universally adopted for chest surgery and is quite suitable for general surgery in the presence of chest disease if secretions are well controlled and respiration is assisted

CHAPTER 32

PHYSIOTHERAPY FOR CHEST DISEASES

JOCELYN M W REED

THE NEED for expiratory breathing exercises for asthmatic and bronchitic patients became widely recognized in about the year 1930 and their value was proved in the asthma research clinics Since then the necessary training in relaxation and in the development of an easy good carriage or posture has been intensified with improvement in results Thoroughness with careful attention to detail are essential for good results in the physical treatment of chest conditions since the movements of the chest are comparatively small and are carried out in a series of joints set at varying angles The patient therefore has no understanding of the capabilities of his chest under voluntary control

The conditions in which the treatment is of a similar type may be grouped together The first group includes bronchitis emphysema and asthma the second comprises pneumonia and pleurisy the third lung abscess collapse and bronchiectasis and the fourth pulmonary tuberculosis

BRONCHITIS EMPHYSEMA AND ASTHMA

Patients suffering from these conditions generally respond best to treatment in groups although the main breathing exercises and particular faults of posture as well as training in relaxation need individual attention and correction which can be given before and after the general part of the class Adult patients derive psychological benefit from finding others in a worse condition than themselves and from hearing of the improvement made by others With children there is scope for imitation and competition in class work

The aims of treatment are the same in these three conditions (1) to gain relaxation (2) to promote breathing control and (3) to develop good posture and balance

Attainment of relaxation

The accessory muscles of inspiration are over toned and tightened to a varying degree The pectorals sternomastoids the upper fibres of trapezius the scaleni and all the adductor muscles of the arm are involved To many patients relaxation of these muscles is difficult or even impossible at first and in bad cases there is a general inability to relax fully any of the large muscle groups of the whole body Those needing a great deal of careful training in relaxation belong to the anxiety type mainly asthmatics They are never mentally and therefore never physically relaxed Some of them cannot keep up with their work and the more they worry the more tense they become

All adult patients and some children need training in general relaxation as well as in local relaxation of their over used muscles of inspiration It is important that a simple explanation of the necessity and importance of relaxation should be

given in a confident and friendly manner before the treatment starts, but when the patient has become accustomed to his surroundings

General relaxation—The patient lies on his back with a pillow under his head, another under his knees, and one under the whole of each arm The arms are slightly bent at the elbows, with the palms of the hands downwards, so that the sides of the thumbs almost touch the body

The patient is shown, the instructor demonstrating with her own arm, the difference between tension and relaxation of the muscles It helps him to understand what is meant by relaxation if he is allowed to hold her arm First the arm is stiffened so that the muscles feel hard, then little by little, the muscles are allowed to relax, so that the patient feels how the arm gradually gets heavier and heavier, until he finally feels its complete relaxed weight Attention is directed in turn to large muscle-groups, starting with those of the hand, forearm, shoulder, neck and so on The patient "talks" to his muscles in his mind telling them gently and frequently, always on a sighing out breath, "let go" This is not a breathing exercise but just natural breathing, which, gradually, will be found to become slower and deeper and more diaphragmatic The patient needs to practise daily for about half an hour at a regular time, and it will be found that the general well being as well as the breathing improves This method is based on a combination of those described by Jacobson (1938) and Fink (1945) It is simple and successful with chest patients when taught with patience and perseverance

Local relaxation—Local relaxation of the tightened inspiratory muscles is gained by rhythmic swinging exercises for the arm and shoulder muscles, and by rhythmic stretching and relaxing shoulder-girdle and spinal movements This will be done during the class part of the treatment

Breathing control

It is not uncommon to find these patients apparently breathing entirely with their upper chests, and for screening to prove that the diaphragmatic movements are poor, sometimes even paradoxical

These breathing exercises are designed first to train relaxation and to control the overworking upper chest, so that it is reduced in size and the stretched lung tissue and bronchial tubes are rested This is done by relaxation training and by slow expiratory breathing exercises

Secondly, they develop in the patient a longer expiratory phase of breathing, to help him in his expiratory difficulty, and to counteract his habit of over inspiration The residual air will thus be reduced Thirdly, the exercises develop fully lower costal and diaphragmatic movements so that the patient can make full use of the bases of his lungs In emphysema, when the patients have raised barrel shaped chests, this procedure is modified They should keep entirely to expiratory breathing without any attempt to gain further expansion

Long expiratory diaphragmatic breathing shown in Fig 184 and expiratory lateral basal expansion exercises shown in Figs 185–186 are the main ones used to develop longer expiration with the fullest use of the lung bases and of the lower half of the chest generally Expiration should last for twice as long as inspiration Good lower costal movement is actually more easily acquired if practised for one



FIG 184—Long expiratory diaphragmatic breathing exercise (a) Full expiration with good contraction of upper abdominal muscles and relaxation of upper chest, (b) inspiration with relaxation of abdominal muscles Note control of upper chest which is still in relaxed position (*By courtesy Institute for Diseases of the Chest*)

(a)



(b)

side separately for a time, as shown in Fig 185, before adding bilateral expansion as in Fig 186

Short expiratory diaphragmatic breathing is used to overcome breathlessness or to prevent or stop an asthmatic attack developing The exercise is described in Fig 187 Positions are arranged so that gravity helps relaxation of the abdominal muscles on inspiration Positions shown in Fig 187 (a) and (b) are used for children and some younger adults It can be modified for adults into a sitting, leaning forward position The position in which the body is in a simple resting posture, slightly rotated forward, is used also for teaching diaphragmatic breathing in difficult cases (Fig 187c)

Control of breathing is practised during strenuous exercise and during ordinary activities such as going upstairs Short expiratory diaphragmatic breathing is

given in a confident and friendly manner before the treatment starts, but when the patient has become accustomed to his surroundings

General relaxation—The patient lies on his back with a pillow under his head, another under his knees, and one under the whole of each arm The arms are slightly bent at the elbows, with the palms of the hands downwards, so that the sides of the thumbs almost touch the body

The patient is shown, the instructor demonstrating with her own arm, the difference between tension and relaxation of the muscles It helps him to understand what is meant by relaxation if he is allowed to hold her arm First the arm is stiffened so that the muscles feel hard, then little by little, the muscles are allowed to relax, so that the patient feels how the arm gradually gets heavier and heavier, until he finally feels its complete relaxed weight Attention is directed in turn to large muscle-groups, starting with those of the hand, forearm, shoulder, neck and so on The patient "talks" to his muscles in his mind, telling them gently and frequently, always on a sighing out breath, "let go". This is not a breathing exercise but just natural breathing, which, gradually, will be found to become slower and deeper and more diaphragmatic The patient needs to practise daily for about half an hour at a regular time, and it will be found that the general well-being as well as the breathing improves This method is based on a combination of those described by Jacobson (1938) and Fink (1945) It is simple and successful with chest patients when taught with patience and perseverance

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FIG 184—Long expiratory diaphragmatic breathing exercise (a) *Full expiration* with good contraction of upper abdominal muscles and relaxation of upper chest (b) *Inspiration* with relaxation of abdominal muscles Note control of upper chest which is still in relaxed position (*By courtesy Institute for Diseases of the Chest*)

(a)

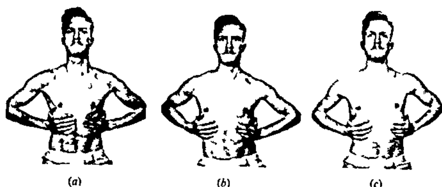


(b)

side separately for a time, as shown in Fig 185 before adding bilateral expansion as in Fig 186

Short expiratory diaphragmatic breathing is used to overcome breathlessness or to prevent or stop an asthmatic attack developing The exercise is described in Fig 187 Positions are arranged so that gravity helps relaxation of the abdominal muscles on inspiration Positions shown in Fig 187 (a) and (b) are used for children and some younger adults It can be modified for adults into a sitting leaning forward position The position in which the body is in a simple resting posture, slightly rotated forward is used also for teaching diaphragmatic breathing in difficult cases (Fig 187c)

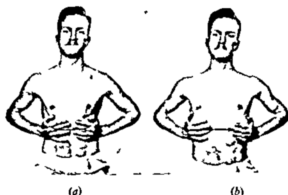
Control of breathing is practised during strenuous exercise and during ordinary activities such as going upstairs Short expiratory diaphragmatic breathing is



(a) (b) (c)

FIG 185 —Unilateral basal exercises for bronchitis and collapse of right lower lobe

left palm



(a) (b)

FIG 186 —Bilateral basal expansion exercise (a) End of slow expiratory phase with pressure of both palms applied in lateral costal area, (b) inspiration with full expansion outwards of both sides against slight palmar pressure

used and when going upstairs is timed to the movements This is first taught on the level

Breathing is arranged rhythmically and slowly, in time with some of the postural muscle strengthening exercises to help breathing control The patient is trained gradually to tolerate more and more exercise without becoming breathless

The psychological approach to the asthmatic patient is slightly different from that to the emphysematous or bronchitic patient These asthmatic patients have to be inspired with confidence in the treatment, and to learn that they can do something themselves to prevent an attack from coming on, instead of having to



(a) (b)



FIG 187—Positions for overcoming breathlessness (a) Contraction of upper abdominal muscles on expiration (quickly) (b) relaxation of abdominal muscles on inspiration (quickly) (c) position for short diaphragmatic breathing exercise

(c)

rely entirely on outside help Much depends therefore on the personality of the physiotherapist The patient is taught to rely on the short expiratory diaphragmatic exercise to prevent the development of an attack or even to stop one that may have developed

Development of good posture and balance

This is the third aim It is achieved by postural muscle strengthening exercises and by developing good posture through particular attention to it during the mobility exercises in class Remedial exercises are given for individual posture deformities

It is essential to develop a well shaped thorax and good carriage otherwise good lower-chest breathing will not be maintained

The patient must stand so that the upper abdominal muscles can be relaxed An imaginary plumb-line dropped from the shoulder should pass through the hip (iliac crest just above the anterior iliac spine) and on down to the forepart of the foot where the weight is carried This makes it possible for the abdominal muscles to be relaxed and thus allow the patient to breathe diaphragmatically

He must learn to carry himself easily and loosely

Constant enquiry into the amount and regularity of practice is essential Asthmatic and emphysematous patients need much encouragement to help them control breathlessness and thus prevent the development of attacks

For all chest patients the vital-capacity estimates, chest measurements and careful assessment of chest movements and postural deformities are made before starting treatment The vital capacity is measured at regular intervals These tests stimulate the patient to further effort as well as being a gauge of improvement

Children with catarrhal bronchitis—For these, training in nose instead of mouth breathing is necessary Limbering up, strengthening postural and balance exercises are needed more than relaxation training Most of the limbering-up exercises are given in the form of rhythmic swinging or " stretch relax " exercises, these bring about relaxation of the tightened accessory muscles of inspiration

Strong remedial exercises, some in kneeling positions and some in crawling positions, are used for bigger children *Good posture is encouraged by the use* of mirrors Later, more difficult balance exercises replace the mirror correction

Other physical treatment

Many of these patients are given a course of general ultra-violet light, administered on alternate days for 20 doses for its general stimulating effect The dose [illegible]

the horny dead layers of cells on the surface Another course may be given after a 2 or 3 months' gap

Short-wave diathermy is beneficial in the treatment of nasal sinusitis partly for its bactericidal effect and partly to produce hyperaemia It can be applied to the chest wall as a form of treatment for asthma, emphysema and bronchitis, in order to increase the circulation

PNEUMONIA AND PLEURISY

Pleurisy

Since pleurisy which does not result from pneumonia is most often of tuberculous or carcinomatous origin, breathing exercises should not be given

Pneumonia

It is most important that breathing exercises should be given early after pneumonia to help re-expand partially collapsed bases, to prevent the development of fibrosis and to expedite resolution Modified exercises can safely be given during broncho-pneumonia, and this is done in many London hospitals and thoracic units with good results

The best form of inspiratory localized breathing exercises are called " pressure expansion exercises " Pressure is used to gain localization of movement and to train the inspiratory muscles by giving them resistance *The exercises are used* to develop full use of the lung bases, particularly on the affected side These exercises are described in detail later, because they are the same as those used for surgical chest conditions

LUNG ABSCESS, COLLAPSED LOBES AND BRONCHIECTASIS

Physical treatment in these conditions is directed to draining the muco purulent secretions arising from the abscess or the bronchiectatic bronchi, and to regaining expansion in those parts of the chest where respiration has been restricted because of the pathological condition

The elimination of muco purulent secretion is facilitated by postural drainage and expiratory breathing exercises, and by tapôtement or percussion treatment consisting in clapping and pressure-vibration

The posture required for successful drainage is decided by consulting the bronchograms It is of importance to find out which bronchi are draining an abscess One or two variations of the apparently anatomically correct posture must often be tried out before good drainage occurs (Fig 188)

Clapping and pressure-vibrations, during long expirations, are the most effective forms of mechanical stimulus to elimination of secretions They are carried out over the affected areas, for bronchiectasis, during the first few minutes of postural drainage For lung abscess clapping should not be allowed, because it might cause haemoptysis, fine vibrations, with a little pressure during prolonged expiration, are used instead In cases in which severe haemoptysis complicates

ed

should be practised in expiratory manner

After this time, if there is still restricted movement, they can be practised in the full inspiratory manner, as described for post-operative surgical patients

PULMONARY TUBERCULOSIS

Patients about to get up after prolonged rest in bed gain considerable benefit from physiotherapy It is particularly useful for "poor breathers", for patients suffering from nervous tension, and in cases of long standing pleurisy

Inadequate respiration

The poor or bad breathers are those who have suffered from breathlessness and who have therefore developed a shallow upper-chest type of breathing This type of respiration is detrimental in patients with bilateral lesions Patients with a low vital capacity who are awaiting thoracoplasty also improve with controlled breathing exercises

Controlled breathing

This controlled breathing consists in careful, gentle, localized development of the area of the chest where the patient has sound lung, combined with training in complete relaxation and control of the lesion areas A patient trained thoroughly and carefully once or twice a day takes between 4 and 8 weeks to get such good control that, when asked to take a deep breath, he only expands the lower chest

The patient with a low vital capacity is trained in diaphragmatic breathing and in careful localized lateral basal expansion of each side, at first separately The vital capacity can usually be raised by 400–700 millilitres in about 3 weeks

Nervous tension

Restless, nervous, worried patients can be helped by training in general physical



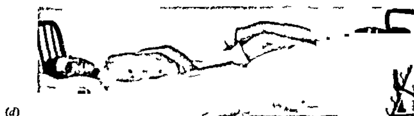
(*a*)



(*b*)

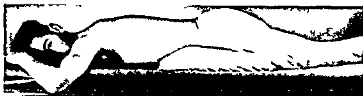


(*c*)



(*d*)

FIG 188—Postural drainage positions (*a*) For drainage of the posterior segment of the right upper lobe (Note the quarter turn or lift backwards [illegible]



(e)



(f)

Fi

(1943) and to practical experience with a large number of cases)

relaxation of the same type as is taught to asthmatic patients The general sense of physical well being induced brings about a release of nervous tension

Pleurisy

Patients with a history of long standing tuberculous pleurisy are sometimes left with a painful stiff chest having restricted movement This condition can be eased by careful localized expansion exercises of the lateral basal area of the affected side, combined with good training in diaphragmatic breathing

Strengthening exercises

Graduated strengthening exercises for all the main anti gravity muscle groups

are important after a long period of rest in bed They should be started weeks before the patient is to get up, so that the strength of the foot, leg, back and neck exercises can be gradually increased This prevents poor posture and flat feet In suitable wards these exercises can be performed in a graduated class

PHYSIOTHERAPY IN SURGICAL CONDITIONS

Pre-operative training in breathing control is of importance in almost all surgical chest conditions, acute empyema being the only exception, but it is in the early post-operative period that localized breathing exercises are essential for the patient's uneventful and speedy recovery In this period post-operative collapse can be prevented by establishing good functioning of the remaining lung or lobes Between 1 and 2 weeks' pre operative training is necessary to establish good control and develop localized expansion movements, unless the patient has a low vital capacity, when 3 or 4 weeks may be necessary

Pre-operative physiotherapy

Pre-operative exercises in chest surgery can be divided into three main groups (1) localized breathing, (2) posture and (3) shoulder movements

Localized breathing exercises

Localized breathing exercises are important to establish breathing control in any cases of thoracotomy for a removal of cysts and non malignant tumours, after empyema drainage or decortication, and in all cases of lung resection

In order to bring about good localized movement of any one part, the rest of the chest must be relaxed Training in physical relaxation is therefore necessary to gain breathing control

In all non tuberculous and non-malignant chest conditions, chest rehabilitation includes the development of full equal bilateral chest movements, and restoration of the ability of each patient to undertake his normal activities and exercise without breathlessness

Postural training

Secondly, a good balanced stance or posture should be taught, as previously described for bronchitic and asthmatic patients This can also be achieved in bed, sitting in a good position, with pillows, arranged to help to maintain it Small corrective active movements should be practised to prevent each possible post-operative fault Pre-operative and post-operative exercises which teach "limbering-up" and postural muscle-strengthening, should be interspersed with the localized breathing exercises

Arm and shoulder movements

Thirdly, arm movements must be maintained by relaxed active shoulder movements taught pre-operatively They include scapular movements with a relaxed arm, gleno-humeral movements with the scapula controlled, and combined shoulder girdle movements The movements are isolated and controlled in order not to stretch the incision and stitches, and to maintain good functioning with minimal physical exertion in the early post-operative period The latter reason is particularly important for tuberculous patients

Postural drainage

In all cases of bronchiectases and lung abscess, intensive pre-operative postural drainage forms the fourth and most important part of their treatment The patient's lung should be as dry as possible before operation, so that post operative collapse, caused by an overflow of secretions into neighbouring bronchi, may be prevented

Localized breathing exercises

Diaphragmatic breathing

Prior to operation, diaphragmatic breathing is of great importance in all types of major chest surgery, because it develops the use of the lung bases and helps to keep them clear of mucus in the early post-operative period Localization of movement to the epigastric and antero lateral lower costal area is taught in conjunction with relaxation and control of the upper chest

During the major part of their pre operative treatment, patients with bronchiectasis and lung abscess are taught the same slow expiratory type of diaphragmatic exercise as used for asthmatic patients, to help in the elimination of the muco-purulent secretions In other surgical conditions, the accent of the exercise is equally on expiration with contraction of the upper abdominal muscles and on *normal diaphragmatic expansion* *The patient should keep his finger-tips resting* lightly on his lower front ribs in order to facilitate consciousness of the movement Actual pressure should never be applied because it produces trick movements For a few days before operation all patients are taught a shortened form, that is the sharp expiratory diaphragmatic breathing used in cases of asthma This is an aid to the elimination of post anaesthetic mucous secretions immediately after the operation, especially in cases of lung resection

Localized costal-expansion exercises

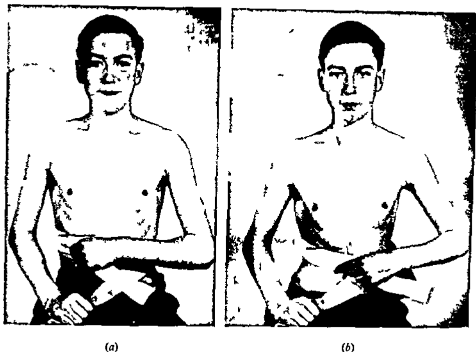
The aim of these exercises is to stimulate good expansion of any areas of the chest where movement is poorly developed or impaired through disease or injury, and at the same time to inhibit other chest movements This inhibition of movement is achieved by relaxation and control, coupled with good expansion of any one part of the chest, it ensures that most of the air inspired will be drawn into the expanding part of the lung

Pressure is applied over the moving costal area in order to focus the attention of the patient on the site and direction of movement required, and, through the resistance given, to develop muscle tone Pressure is applied at first by hand and later by using a belt

For these purposes the chest is divided into four main areas (1) *The lateral basal area*, in which expansion of the chest is outwards against pressure applied by the palm or knuckles on the side of the lower ribs (For position of hands *see* Fig 185 Figure 189 shows the pressure applied by the belt)

(2) *The upper lateral area*, in which expansion of the chest is outwards against pressure applied by either the palm of the hand or the fingers of the opposite hand, placed high on the side (Fig 190 *a* and *b*)

(3) *The pectoro-apical area*, in which expansion of the chest is upwards and forwards against pressure applied by the palmar surfaces of the fingers, placed



(a) (b)

Fig 189 —T W (age 17 years) has had a right middle and lower lobectomy for bronchiectasis He is having localized basal expansion exercise (a) breathing in while expanding lower ribs against the belt (b) breathing out showing the belt pulled firmly prior to next expansion

over the upper chest The tips of the fingers rest just below the clavicle the opposite hand being used (Fig 190 *c* and *d*)

(4) *The posterior basal area* in which expansion of the chest is mainly backwards against pressure applied by a belt placed across the back, covering the upper posterior basal area

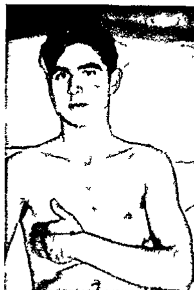
These localized costal exercises are practised pre operatively in areas of the chest where movement is poorly developed or has become restricted and after operation when it is desired that full expansion shall be maintained or regained As soon as movement has improved on the weak side to about three quarters of that on the sound side, bilateral movement is practised This is necessary to develop the normal co ordinated movements of both sides of the chest

The method of applying pressure is first taught by the physiotherapist by applying it herself It is then practised by the patient with his own manual pressure Later a belt is used for lower and upper costal expansion exercises because the shoulders can be more relaxed The belt crosses in front the piece coming from the left side being held by the right hand and that from the right side by the left hand (Fig 189)

Localized costal exercises can be practised in an expiratory manner or in an



(a)



(b)



(c)



(d)

FIG. 100 [illegible] removal of ne[illegible]rofibroma (right upper [illegible]

inspiratory manner In the former case expiration takes at least twice as long as *inspiration and it is accompanied* by extra pressure towards the end of the expiratory phase The exercises are given in this manner prior to operations for bronchiectasis and lung abscess and in cases of asthma and emphysema The inspiratory manner is used for empyema after thoracotomy, and after lobectomy and pneumonectomy It is also used to stimulate the expansion of collapsed lobes and in convalescence from pneumonia

When good expansion is required the patient is taught to breath in with as strong expansion as possible against applied pressure maintain the expansion while holding the breath and finally relax while breathing out This is followed by a pause before repeating the movements

Each of the three phases are of equal duration The middle phase however, should not be allowed if a fistula is present

Physiotherapy after thoracotomy

Physiotherapy is needed after thoracotomy performed to treat empyema or for removal of tumours cysts *neurofibroma or foreign bodies (when lung resection* is not performed) or for ligation of a patent ductus arteriosus

Breathing exercises—Breathing exercises should be practised carefully for 1 2

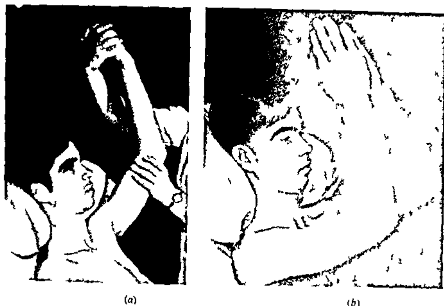
expansion exercises of the full *inspiratory type* are taught for each area of the affected side of the chest Normal diaphragmatic breathing and bilateral basal expansion are practised to develop full use of the lung bases

Postural exercises—Simple posture corrective movements are taught to help hip, spine *and shoulder alignment These movements are important while the* patient is in bed but as soon as possible graded class exercises for muscle strengthen ing and posture training replace them

Physiotherapy before and after pulmonary resection

Surgical conditions other than tuberculosis or carcinoma

In cases in which lobectomy or pneumonectomy is performed for diseases other than tuberculosis or carcinoma pre operative treatment includes a course of 3–5 weeks postural drainage with tapotement or percussion massage This consists in clapping (except when there is a history of haemoptysis) and of pressure vibrations applied during prolonged expiration These manipulations should be carried out alternately for the first 3 5 minutes of each tipping session Classes should be held daily for limbering up localized expiratory breathing exercises and good posture training During the week before the operation the patient is taught short sharp expiratory diaphragmatic breathing and localized costal expansion exercises *in the full inspiratory manner for the remaining sound lobe* or lobes which will be needed in the immediate post operative phase In pre paration for lower lobe lobectomy, expansion exercises are practised in the



(a) (b)

FIG 191 —Same patient as shown in Fi 90 [illegible]

[illegible] to the affected side

pectoro apical and upper lateral areas In the immediate post operative phase good expansion of the upper lobe should be maintained In 3–4 days when the skiagrams show that there is full upper lobe expansion lateral basal expansion is started to expand the upper lobe downwards By the end of 2 3 weeks expansion should have increased so that there is no space between the upper lobe and the diaphragm

For patients undergoing middle lobe lobectomy since the lower lobe readily tends to collapse mainly posterior and lateral basal expansion exercises should be practised in the pre operative and immediate post operative phases

For patients submitted to pneumonectomy although all areas of the affected side should be well exercised beforehand lateral basal and upper lateral costal expansion of the sound side and diaphragmatic breathing are the important exercises When these movements are well developed about a week after operation expansion movements on the side of the pneumonectomy are practised to prevent the development of scoliosis Bilateral expansion exercises for the lower and middle-chest develop normal co ord nated movements

Operations for tuberculosis or carcinoma

When lobectomy or pneumonectomy is performed for tuberculosis or carcinoma

inspiratory manner In the former case expiration takes at least twice as long as inspiration and it is accompanied by extra pressure towards the end of the expiratory phase The exercises are given in this manner prior to operations for bronchiectasis and lung abscess and in cases of asthma and emphysema The inspiratory manner is used for empyema after thoracotomy and after lobectomy and pneumonectomy It is also used to stimulate the expansion of collapsed lobes and in convalescence from pneumonia

When good expansion is required the patient is taught to breath in with as *strong expansion as possible against applied pressure* maintain the expansion while holding the breath and finally relax while breathing out This is followed by a pause before repeating the movements

Each of the three phases are of equal duration The middle phase however should not be allowed if a fistula is present

Physiotherapy after thoracotomy

Physiotherapy is needed after thoracotomy performed to treat empyema or for removal of tumours cysts neurofibroma or foreign bodies (when lung resection is not performed) or for ligation of a patent ductus arteriosus

Breathing exercises —Breathing exercises should be practised carefully for 1-2 weeks pre operatively (except for empyema) and started within 24 hours of the operation so that the lung may be re expanded before the pleura has time to become thickened or fibrous scar tissue and adhesions can be formed Localized costal expansion exercises of the full inspiratory type are taught for each area of the affected side of the chest Normal diaphragmatic breathing and bilateral basal expansion are practised to develop full use of the lung bases

Postural exercises —Simple posture corrective movements are taught to help hip spine and shoulder alignment These movements are important while the patient is in bed but as soon as possible graded class exercises for muscle strengthen ing and posture training replace them

Arm and shoulder movements —Relaxed active shoulder movements are changed to resisted movements and free exercises as soon as possible (Fig 191) Elevation *of the arm should be of full range by the fifth or sixth day*

Physiotherapy before and after pulmonary resection

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(a)



(b)

Fi

arm on the affected s de

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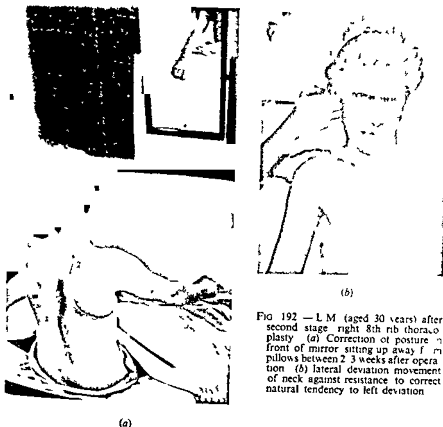
Postural exercises—Simple posture corrective movements are taught to help hip, spine and shoulder alignment These movements are important while the patient is in bed, but as soon as possible graded class exercises for muscle strengthening and posture training replace them

Arm and shoulder movements—Relaxed active shoulder movements are changed to resisted movements and free exercises as soon as possible (Fig 191) Elevation of the arm should be of full range by the fifth or sixth day

Physiotherapy before and after pulmonary resection

Surgical conditions other than tuberculosis or carcinoma

In cases in which lobectomy or pneumonectomy is performed for diseases other than tuberculosis or carcinoma, pre-operative treatment includes a course of 3 5 weeks postural drainage with tapotement or percussion massage This consists in clapping (except when there is a history of haemoptysis) and of pressure-vibrations applied during prolonged expiration These manipulations should be carried out alternately for the first 3-5 minutes of each tipping session Classes should be held daily for limbering up, localized expiratory breathing exercises and good posture training During the week before the operation, the patient is taught short sharp expiratory diaphragmatic breathing and localized costal expansion exercises in the full inspiratory manner for the remaining sound lobe, or lobes, which will be needed in the immediate post operative phase In pre paration for lower lobe lobectomy, expansion exercises are practised in the



(a)

(b)

Fig 192 — L M (aged 30 years) after second stage right 8th rib thoracoplasty (a) Correction of posture in front of mirror sitting up away from pillows between 2 3 weeks after operation (b) lateral deviation movement of neck against resistance to correct natural tendency to left deviation

of the long incision, the postural and arm movements are isolated to a single movement at a time

All the factors concerned with the maintenance of good posture are demonstrated with the use of a mirror placed at the foot of the bed (Fig 192a—here the patient is seen 3 weeks after second stage thoracoscopy) Corrective movements are practised for any present faults and to prevent those likely to occur after operation (Fig 192b—here neck correction is being practised 3 weeks after right second stage thoracoscopy)

Diaphragmatic breathing is practised in the patient's own time and with complete relaxation of the upper chest This is the most important breathing exercise, ensuring good use of the base of the lung on the thoracoplasty side and helping to prevent paradoxical breathing in the upper chest after operation

Localized postero-lateral basal expansion is practised on the side for thoracoplasty A little pressure is applied to help the patient to feel the movement, but it is applied gently so that it is localized This exercise is essential for patients

pre operative physical treatment consists in 1–2 weeks' *individual training in relaxation and control of the chest, coupled with diaphragmatic breathing and with small localized unilateral expansion movements of the healthy parts of the lungs* In tuberculous cases the amount of good diaphragmatic movement that is possible will depend on whether or not there has been a phrenic avulsion or a pneumoperitoneum is present Relaxed active shoulder movements and postural corrective exercises are practised before operation but they are of *secondary importance to the controlled breathing exercises*

In preparation for upper lobectomy, small pectoro apical expansion movements are practised on the sound side for a few days if there is a large cavity in the upper lobe on the affected side *If there is infiltration without cavity formation in the upper lobe, this pectoro apical expansion can be practised gently on the affected side* After operation, this exercise and upper lateral costal expansion are used to help to expand the lower lobe upwards As soon as expansion is complete (in about 2 weeks) breathing exercises are stopped Postural corrective movements and graduated muscle strengthening exercises may be needed for a further 2 weeks and should be supervised until the patient is ambulant *Foot and leg exercises should be practised for 2 weeks before the patient gets up*

When pneumonectomy is performed for carcinoma the patients tend to be elderly, to have stiff chests, and to use the upper parts excessively In the short pre operative period good diaphragmatic movement and unilateral lower costal expansion of the healthy side are developed *If these breathing exercises are practised frequently in the early post operative phase, there is little danger of collapse of the lower lobe, or of pneumonia caused by infection of retained secretions* Graduated *foot and leg exercises should be started on the day after* [illegible] These patients usually begin [illegible] exercises are continued and [illegible]

patients

When pneumonectomy is performed on account of tuberculosis, similar breathing exercises are used but the foot and leg exercises are postponed until a later date Postural corrective movements need more attention with younger *and more mobile patients, some of whom have poor postural muscle tone*

Physiotherapy for plastic operations

In these operations there are extensive muscle incisions and rib resections, and *therefore postural muscle strengthening and training are essential*

Thoracoplasty

Pre operative exercises—Many of the patients who undergo thoracoplasty have poor muscle tone and posture resulting from long periods of rest in bed and of curtailed physical exertion Some have a shallow upper chest type of respiration with low vital capacity Pre operative training is needed for between 1 and 3 weeks *Care should be taken to graduate these movements and exercises in order* not to tire the tuberculous patient For this reason, and to prevent any stretching



(a)



(b)

FIG 192 — L M (aged 30 years) after second stage right 8th rib thoracoplasty (a) Correction of posture in front of mirror sitting up away from pillows between 2 3 weeks after operation (b) lateral deviation movement of neck against resistance to correct natural tendency to left deviation

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Localized postero lateral basal expansion is practised on the side for thoracoplasty A little pressure is applied to help the patient to feel the movement, but it is applied gently so that it is localized This exercise is essential for patients

undergoing general anaesthesia Training in relaxation of shoulder and arm muscles is needed, so that movements can be isolated, so far as is possible, to individual muscle-groups Movements of the scapula, with relaxed arm-depression and retraction, and gleno-humeral movements with controlled scapula are taught Those most used are elbow raising to shoulder level in different planes to abduct the arm, extension, and outward rotation

All shoulder-joint movements are performed with a bent elbow, in order to give the patient a small weight to lift and therefore the least strain on the muscles controlling the scapula The arm is elevated above the head, with assistance at first (Fig 191*a*), and later, when the movement is practised by the patient, both arms are raised, the palms meeting (Fig 191*b*) Ultimately he can separate the arms above the head, and practise raising the affected arm by itself

Post operative exercises—After the second-stage operation, when the ribs beneath the scapula have been removed, elevation of the arm should not be practised for 5–7 days Until the scapular movements are strong, elevation is painful, and it can shift the scapula when it has been placed forwards with its inferior angle beneath the seventh and eighth ribs

In the event of post-operative collapse of the lower lobe, postural drainage is arranged for half an hour 3 times a day for a few days The patient is placed on his sound side with a pillow support for his head and neck, which should not be allowed to drop towards the sound side The foot of the bed is raised 16 inches from the floor Diaphragmatic and postero-lateral basal expansion exercises are practised with expiratory emphasis

Postural muscle-strengthening exercises should be continued until the patient is allowed up, and then should be supervised once or twice a week either in ward classes or individually Foot and leg exercises are started in the last weeks prior to getting up When the patient is up, classes in graduated posture training, walking and balance exercises prevent postural deformities arising from bad habits of stance

Extrapleural pneumolysis

In cases of extrapleural pneumolysis, the postural back muscle incisions are nearly as extensive as in thoracoplasty There is, therefore, a similar, though less severe, temporary imbalance of the upper thoracic extensor and shoulder retractor muscles Pre operative training in localized postural corrective movements and diaphragmatic breathing, with relaxation of the upper chest, is practised as for thoracoplasty patients Usually about 4 periods are sufficient Because in such cases the tuberculosis is frequently more active, precautions should be taken not to tire the patient Localized arm movements can be practised once or twice with the opposite arm Post operative treatment is not usually necessary for more than 2 weeks

[illegible] ...fts are used for ... the graft ... deltoid can be practised to prevent atrophy, but neither stretching nor contraction of the muscle used for the graft is allowed for the first 14 days Movements are then gradually increased The arm is either kept in a sling or lightly bound to the side, to

prevent the patient from using it inadvertently Postural corrections and breathing exercises are used

Posture

In the physical rehabilitation of 'surgical chest' patients, co-operation between nurses and physiotherapist is necessary to maintain good posture in bed and to ensure early coughing to eliminate post anaesthetic secretions and thus prevent collapse of the lower lobes

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